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LXIX

LYMPHANGIOMA OF THE TONSIL

REPORT OF A CASE WITH A CRITICAL REVIEW OF THE LITERATURE

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To our knowledge, the first reported case of a benign tonsillar neoplasm was that of Roberts[1] in 1827, and the first complete monograph on the subject of benign tumors of the tonsil was that of Ardenne[2] in 1897. In a review of the literature since then, one is impressed not only with the fact that benign tumors of the tonsil are uncommon but also that vascular tumors in general are rare.

Most angiomatous lesions of the tonsil reported in the literature are hemangiomas, but a few are lymphangiomas. New and Childrey[3] in 1931 reported only 63 cases of benign tumors of the tonsil and pharynx seen at the Mayo Clinic between 1917 and 1930. Five of these tumors were vascular in nature—two varices, two hemangiomas, and one lymphangioma—but all of these five occurred in the pharynx exclusive of the tonsil. Caliceti[4] in 1923 collected 107 cases of benign tonsillar neoplasms, of which 12 were called "angiomas" and one a

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fibroangioma. Ormerod[5] in 1926 reported a benign vascular tumor of the tonsil, to which he applied the non-committal name "angioma." At that time, he found 19 reported cases of hemangioma and 3 cases of lymphangioma of the palatine tonsils in the world literature. Fraenkel[6] in 1933 described a case of "cavernous hemangioma" of the tonsil. He reviewed the literature at that time and found four cases of hemangioma of the tonsil that had been reported since Ormerod's review, those of Trible 1927, Diaz 1928, Pinaroli 1928, and Resnikow 1927.

A few reports are found in the literature in which the surgeon described an angiomatous lesion of the tonsil but did not subject the lesion to pathologic study. Such a case is that of Herbert,[7] who in 1924 described a "varix" of the upper pole of the left tonsil. During the tonsillectomy, however, the "varix" bled profusely, and he chose to leave the lesion in the patient. Garelli[8] also described two cases of cavernous hemangioma, which he saw clinically but which he did not biopsy. One of these lesions was limited to the right tonsil, but the other was a part of a more extensive cavernous hemangioma involving the skin of the face and the mucous membrane of the cheek and palate.

Not infrequently in the mucous membrane of the nose and throat of young people, fibromatous tumors with telangiectatic capillaries or lymphatics are found. These have been called angiofibromas or fibroangiomas. Brill[9] and Hara[10] both report cases of such a tumor isolated to the tonsil. These angiofibromas, however, should not be confused pathologically with true tumors of blood and lymph vessels. It is this latter group that is being discussed in this paper.

It is difficult to assess the true relative incidence of hemangiomas and lymphangiomas of the tonsil for several reasons. First, many authors have been satisfied merely to classify their cases as angiomas and have made no attempt to subdivide them into either lymphangiomas or hemangioma types. An example of this is that of Ormerod.[5] He reported his case as simply an angioma of the tonsil, but the accompanying photomicrograph is strongly suggestive of a lymphangioma. We are prevented from more specifically categorizing other cases of "angiomas" of the tonsil, such as that of De Francesco,[11] because the author does not give a microscopic description or a photomicrograph of the tumor. A second reason for difficulty in ascertaining the actual incidence of hemangiomas and lymphangiomas of the tonsil is caused by the different classifications used by different authors. An example is Liveriero's[12] case, which he called a "telangiectatic

fibroma" of the tonsil. The photomicrograph of this tumor, however, looks like a lymphangioma.

Because we believe that some lymphangiomas of the tonsil have been simply classified as angioma and others classified under misleading names, it is the opinion of the authors that lymphangioma of the tonsil may not be as rare as a superficial appraisal of the literature would lead one to think. We also believe that anatomic factors may favor a higher ratio of lymphangiomas to hemangiomas than is reported in the literature. The tonsil is a lymphoid organ and as such it is not only rich in lymphocytes and reticuloendothelial cells but also in efferent lymphatics.

The occasion for this review and critique of the literature on lymphangiomas of the tonsil was prompted by a recent case, which the authors had the opportunity of studying in the Department of Otolaryngology and the Laboratory of Surgical Pathology of the Columbia-Presbyterian Medical Center. We would like to present a clinical history of this case and briefly compare it with that of Menzel,[14] the only other lymphangioma of the tonsil available for us to review. To our knowledge, this is the first case of lymphangioma of the tonsil reported in the United States and the fourth reported as a lymphangioma of the world literature. The previous well-documented cases are those of Goris[13] * and Menzel.[14] Another case is mentioned by Ormerod[5] without referring to either author or journal.

REPORT OF A CASE

This patient, a 27-year old white Cuban housewife, was first seen in the Ear, Nose and Throat Clinic on February 10, 1960, complaining of a progressive sore throat over a period of two weeks. The throat soreness was accompanied by daily episodes of "spitting up blood" which was reported to be of small quantities. The patient had recently felt somewhat weaker, and has had occasional chills and right neck pain. A vague history of hearing loss was elicited, and tinnitus was claimed to be present in the right ear. Antibiotic therapy was given by the patient's physician two days prior to the hospital visit.

Past medical history revealed an appendectomy at age 16, cholecystectomy at age 23, two miscarriages, and recent therapy for thyroid

* The journal in which this case was reported was not available to us.

Fig. 1.—In-situ illustration of lymphangioma of right tonsil (copied from a technically limited photograph).

dysfunction. The patient denied any allergies or systemic bleeding tendencies.

Physical examination presented a well-developed, moderately obese white female appearing to be in good general health. Blood pressure 100/60, pulse 80, respiratory rate 20, and temperature 99.2° F.

Examination of the head, ears, and nose was not remarkable. The oral cavity and oropharynx presented an irregular, nodular, bluish enlargement of the right tonsil, strictly limited by the anterior and posterior pillars plus the base of the tongue (Fig. 1). Several fresh bleeding points were noted in the mid-surface of this tumor. Pulsations were neither visualized nor palpated. The consistency was soft. Also noted, were several bluish punctate areas in the right pterygomandibular raphe. The left tonsil was normal in appearance.

Further examination disclosed a diffusely enlarged thyroid gland, and a fullness of the supraclavicular fossae, which contributed to the truncular type of obesity of this patient. The remainder of the physical examination proved not to be revealing.

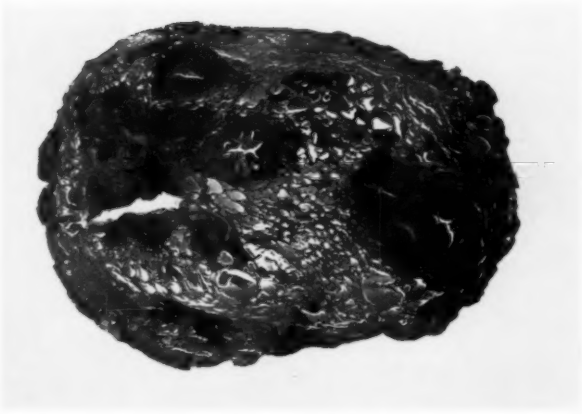

Fig. 2.—Low-power view of the right tonsil and the tumor (x9).

Laboratory studies were carried out in an attempt to determine the extent of the pathology and to rule out bleeding tendencies and endocrinopathy. X-rays of the skull, chest, and abdomen were within normal limits. A complete blood count, including a platelet count, were within normal range. Studies of the coagulation mechanism included prothrombin time, venous clotting time, bleeding time, and clot retraction time. All these tests failed to show any abnormalities and performance of the capillary fragility test did not produce petechiae. Although the "buffalo type" of obesity was striking in this patient, the fasting blood sugar, urinary 17-ketogenic steroids, 17-ketosteroids, and serum alkaline phosphatase values were such that the presence of Cushing's disease or Cushing's syndrome was doubtful. The protein bound iodine level was normal, and the radioactive iodine uptake was normal. No pathogens were found on throat culture. Audiometric testing did not show a hearing loss. A carotid angiogram was omitted, as it was felt that the pathology most probably did not extend beyond the tonsil.

Eight days following hospital admission, a tonsillectomy was performed under general anesthesia. The tumor and the left tonsil were easily removed by the dissection technique and hemostasis was accomplished by several ligatures in the right fossa. There was no postoperative bleeding, and discomfort was not excessive. The patient was seen on the second and fifth postoperative day, revealing an uncomplicated recovery.

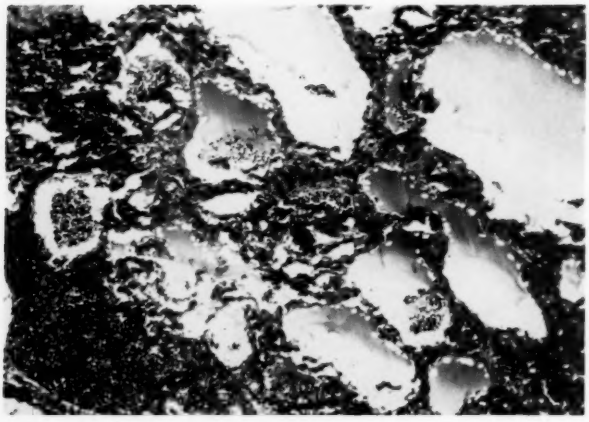

Fig. 3.—Demonstrates the lymphatic channels of the tumor within the rich lymphoid tissue of the tonsil (x120).

Fig. 4.—Lymph channels growing between the skeletal muscle fibers of the tonsillar fossa (x120).

GROSS AND MICROSCOPIC DESCRIPTION OF THE TUMOR

The surgical specimen as it appeared in the Laboratory of Surgical Pathology of the Columbia-Presbyterian Medical Center consisted of two parts. The first part, labelled right tonsil, consisted of an ovoid, firm, 1.4 x 0.8 x 0.8 cm piece of tissue with a glistening, somewhat purplish irregular surface, which in areas demonstrated a lobulated, grape-like appearance. The second part, labelled left tonsil, consisted of an ovoid, smooth-surfaced 1.0 x 0.8 x 0.7 cm piece of tissue with a homogeneous pinkish appearance.

Microscopic sections of the left tonsil revealed well preserved tonsillar architecture with deep crypts extending into lymphoid follicles. The right tonsil, however, showed extensive replacement of the normal tonsillar architecture by a tumor composed of closely-packed, large endothelial-lined spaces of varying calibre filled with an eosinophilic homogeneous fluid (Fig. 2). Delicate fibrous trabeculae separated the lymph channels, and the tumor stroma was rich in lymphoid tissue (Fig. 3). The tumor extended into the skeletal muscle fibers of the tonsillar fossa (Fig. 4). This extension was not considered to be true invasion, however, and in none of the sections was evidence of malignancy recognized.

The tumor is considered to be a lymphangioma rather than a hemangioma for two reasons. First, because the neoplastic vessels are associated with a rich lymphoid stroma, and second, because in almost none of the vessels are formed blood elements found. The occasional vessel containing blood elements may have been the result of a rupture of a neoplastic lymphatic channel with resulting passage of blood into the vessel. This also would explain the clinical symptom of bleeding. The basis for the blue appearance of the tumor grossly cannot be stated. However, it may have been the result of seeing the tonsillar lymphoid tissue through the translucent, dilated, lymphatic channels of the tumor.

SUMMARY

In this paper, the authors have critically reviewed the literature of angiomatous lesions of the tonsil, with special emphasis on lymphangiomas, and have presented a recently observed case of lymphangioma of the tonsil, which presented with signs and symptoms of bleeding from the mouth and sore throat and which was easily treated by tonsillectomy. The symptoms and the pathologic findings in this case were very similar to those presented in the only other case report

available to us (Menzel[11]). To our knowledge, this is the first reported case in the United States.

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The authors wish to express their gratitude to Drs. R. Lattes and E. P. Fowler, Jr., for their helpful criticism and suggestions.

REFERENCES

1. Roberts: A Case of Papilloma of the Tonsil. Arch. Otol. 25:55-57, 1896, New York.

2. Ardenne, J. F.: Tumeurs Benignes de L'Amygdale, Paris, J. B. Bailliere and fils, 1897.

3. New and Childrey: Tumors of the Tonsil and Pharynx. Arch. Otol. 14: 596-609 (Nov) 1931.

4. Caliceti, P.: Bollettino di Malattie d'Orrechio, 1923.

5. Ormerod, F. C.: Angioma of the Tonsil. J. Laryn. and Otol. 41:797-800 (Dec.) 1926.

6. Fraenkel, I.: Tonsillencavernom. Zeitschrift Fuer Hals, Nasen, und Ohrenheilkunde 33:268-274, 1933.

7. Herbert, W.: Varix Complicating Tonsillitis. Laryngoscope 34:50-51 (Jan.) 1924.

8. Garelli, F.: Angiomas of Mouth and Tonsils. Miverva Chirurgica 6:93-96 (Feb.) 1951.

9. Brill, J.: Angiofibroma Tonsillae. Zeitschrift Fuer Laryngologie und Rhinologie 16:62 (July) 1927.

10. Hara, H.: Benign Tumors of the Tonsil. Arch. Otol. 18:62-69 (July) 1933.

11. DeFrancesco, D.: Interesting Angiomas of the Mouth and Tonsils. Arch. Ital. di Otol. 48:607-613, 1936.

12. Liveriero, E.: Contributions to the Study of Benign Tumors of the Palatine Tonsils. Ann. di Laringol., Otol., Rinol., Faringol., Vol. (New Series) 7-8, 1932.

13. Goris: Cavernous Lymphangioma of the Tonsil. Ann. Belgian Soc. Surg., 1910.

14. Menzel: Lymphangioma of the Left Tonsil. Monatschr. der Ohrenheil. 53: 509, 1919.

LXX

THE EFFECTS OF ANESTHETICS UPON THE EAR
II. PROCAINE HYDROCHLORIDE

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In an earlier paper[7] it was pointed out that the use of topical anesthetics is considered by many surgeons to be the anesthetic method of choice for operations on the ear. Pitkin[6] indicated the favorable conditions accompanying topical anesthesia, and Goodman and Gilman[4] report that the great practical value of local anesthetic drugs arises from the fact that their action is reversible and is followed by complete recovery of nerve function with no evident structural damage to nerve fibers or cells. Nevertheless, evidence now points to the fact that certain currently used local anesthetics produce irreversible losses in the cochlear response when these solutions are employed to anesthetize middle ear structures during surgery. According to the electrical hypothesis of auditory nerve stimulation, such losses in the magnitude of the cochlear potential cannot help but have deleterious effects upon hearing. It is possible that while local anesthetics do not cause structural damage to nerve fibers or cells these solutions apparently cause serious damage to the delicate sensory structures of the cochlea. Accordingly, the choice of anesthesia for otological surgery is an important matter, and one which needs very careful study.

Rahm, Strother, Gulick and Crump[7] have demonstrated that the application of cocaine (10 per cent) and hexylcaine hydrochloride (Cyclaine®) (5 per cent) to the round window membrane of the

From the Princeton Psychological Laboratory. This work was supported by a contract between the National Institutes of Health and Princeton University NIH B-2125, and by Higgins funds allotted to Princeton University. Permission is granted for reproduction and use by the United States Government.

cat produced serious losses in the magnitude of the cochlear responses which resulted eventually in its total abolition; while a 1 per cent solution of hexylcaine chloride only temporarily reduced the cochlear response.

Other experimenters have studied the effects of various chemical substances upon the electrical response of the cochlea.[2,3,8] This literature is reviewed elsewhere.[7,8]

The present experiment studied the effects of procaine hydrochloride (2 per cent) upon the cochlear response of the cat. It is customary to use procaine hydrochloride as a reference for measuring the potency of anesthetics, and it was deemed advisable to obtain data on the effects of this drug upon the ear so that comparisons might readily be made between it and selected anesthetics currently in use for middle ear surgery.

PROCEDURE

Cats were used as experimental animals, and each was anesthetized with a solution of diallyl-barbituric acid and ethyl carbamate injected intraperitoneally in a dosage of 1 cc per kilogram of body weight. After a surgical level of anesthesia had been reached, the tympanic bulla was approached laterally through an oval incision in the skin covering the superficial facial vein. The vein was ligated and the parotid gland was removed. Resection of the sternocleidomastoid, digastric, and stylohyoid muscles exposed the auditory bulla.

One hole (diameter 6 mm) was drilled in the lateral surface of the bulla. This hole accommodated the wire from the active electrode which terminated on a piece of platinum foil placed on the round window membrane. In addition, the hole was used to gain access to the round window for the application and removal of procaine hydrochloride and control solutions of normal saline.

A tonal stimulus of 1000 cps was generated by a General Radio, Type 913-C beat-frequency oscillator whose output was connected to a Daven, Type T-693 attenuator. The output from the attenuator fed into a power amplifier which drove a Western Electric 555 receiver. A hard rubber tube which terminated in a cannula tied into the external meatus led aerial sound from the speaker to the tympanic membrane. Cochlear potentials picked up from the round window membrane were amplified and measured with a General Radio, Type 736-A wave analyzer operated as a selective voltmeter.

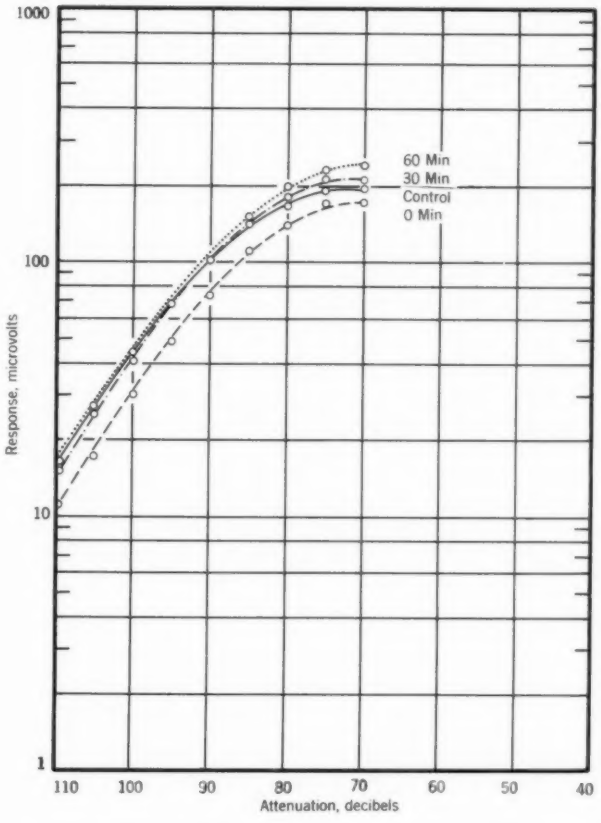

Fig. 1.—Intensity functions for a representative cat treated with procaine hydrochloride (2 per cent) applied to the round window membrane.

The intensity of the tonal stimulus was adjusted initially so as to give a cochlear response of 50 microvolts. After the stability of the response was assured, either normal saline or procaine hydrochloride was applied to the round window membrane by means of a small nylon tube affixed to a hypodermic needle on a 1 cc syringe. The temperature of these solutions equalled the body temperature of the preparation. The blunt end of the tube, placed in the bulla cavity, was located directly over the round window membrane at a distance of about 2 mm. The dosage was always 0.25 cc. After 15 minutes

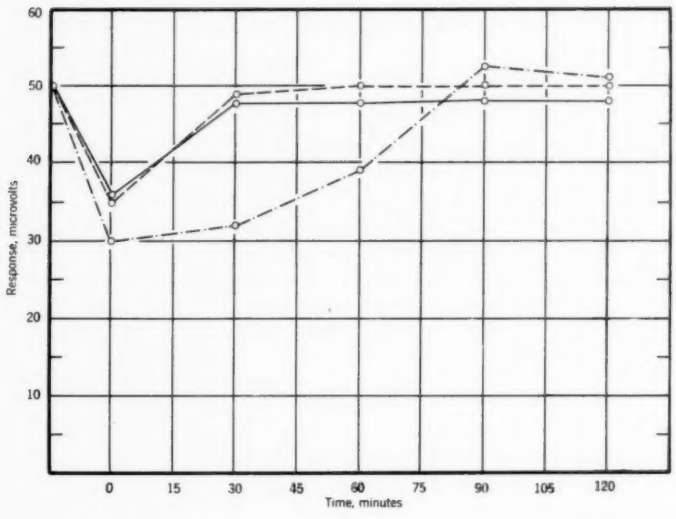

Fig. 2.—Comparisons of three cats treated with procaine hydrochloride (2 per cent).

the substance was removed by using the nylon tube as a catheter. In addition, the membrane was dried with a cotton wick. During removal of the procaine hydrochloride and normal saline, the active platinum electrode was removed from the round window membrane.

The effects of procaine hydrochloride and normal saline upon the cochlear response were evaluated in two ways. First, changes in the magnitude of the response produced by a constant stimulus were observed over extended periods of time. Second, intensity functions were plotted immediately before and after application, as well as at regular intervals following withdrawal. All animals were isolated in an electrically shielded, soundproof room during the experiment.

RESULTS

Application of either normal saline or procaine hydrochloride to the round window membrane resulted in an immediate and precipitous drop in the magnitude of the cochlear response. This drop may be ascribed to the electrical shunting effect of the solutions on the coch-

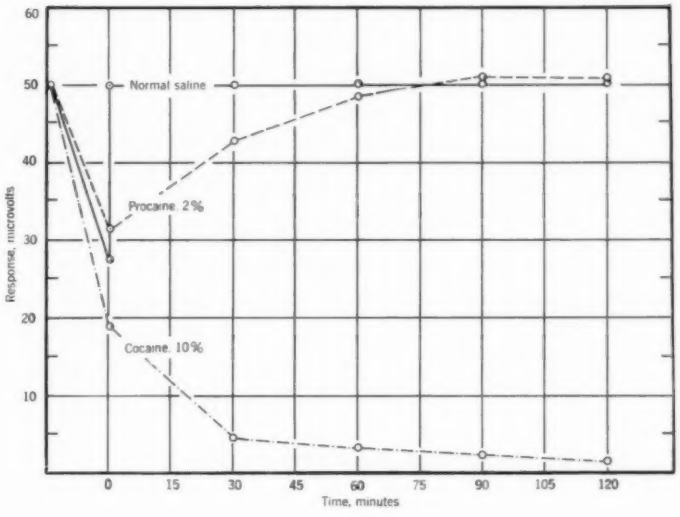

Fig. 3.—Comparison of the effects of normal saline, procaine hydrochloride (2 per cent) and cocaine (10 per cent) upon the cochlear response.

lear potential at the round window. Further changes in the cochlear response depended upon the particular solution applied.

Normal Saline. After saline had been withdrawn and the round window membrane had been dried, the response recovered fully and immediately. It remained stable as long as the preparation remained in satisfactory condition. No changes were observed in the intensity functions obtained before and after treatment with saline.

Procaine Hydrochloride (2 per cent). Figure 1 presents several intensity functions obtained from a representative cat. The cochlear response is given in microvolts, and the intensity of the stimulus is given in decibels (96 db equals 1 dyne per square centimeter). The function obtained prior to the application of procaine hydrochloride (control) shows the typical linear relationship between the intensity of the stimulus and the magnitude of the cochlear response. The other functions show the temporary loss in response immediately after withdrawal at 0 minutes and at 30 and 60 minutes following withdrawal. Recovery was complete within 30 minutes of withdrawal, and the

response remained stable during the next two hours, at which time the experiment was terminated.

Data obtained on three other animals are presented in Figure 2. Here the effects of procaine hydrochloride upon the cochlear response are evident: procaine hydrochloride reduced the response, but complete recovery followed. The drop in the magnitude of the response produced by procaine hydrochloride was small (about 4 db), and complete recovery took between one-half and one and one-half hours.

COMMENT

The electrical shunting effect observed during the 15 minute period of application made it impossible during the 15 minute interval to determine the effects of the normal saline and procaine hydrochloride upon the cochlear response. However, the effects became clear with the removal of these solutions.

As shown in Figure 3, the normal saline had no effect except electrical shunting. When the round window membrane was dried and the electrode replaced, the response magnitude equalled the pre-application level. This immediate recovery demonstrates that the general procedure employed did not introduce any conditions which of themselves produced losses.

The effects of procaine hydrochloride also are summarized in Figure 3. The curve is based upon data obtained on the three preparations shown in Figure 2. At the time of withdrawal of this anesthetic the drop in response was about 4 db. Unlike the recovery following treatment with saline, in these preparations the response recovered more slowly. This was probably due to the effects of procaine hydrochloride upon the hair cells of the organ of Corti.

For purposes of comparison, data obtained in an earlier study[7] in which cocaine (10 per cent) was applied to the round window are also presented in Figure 3. The comparison of procaine hydrochloride and cocaine reveals two important differences in the effects of these anesthetics upon the ear. First, the drop in response which occurred during the period of application was more severe for cocaine (about 8 db) than for procaine hydrochloride (about 4 db). Second, on each of four preparations treated with cocaine the response not only failed to show any recovery but actually continued to decrease until it disappeared. The application of procaine hydrochloride (2 per cent) was always followed by complete recovery.

It is known that permanent damage to tissues may result from improperly prepared solutions of anesthetic substances. Aqueous solutions of salts of local anesthetic drugs may exert an osmotic pressure more or less than that of tissues depending upon their strength. Hyper- or hypotonic solutions disturb the osmotic equilibrium between the cells and their external environment.[1] The present study made use of standard commercially prepared anesthetic solutions.

Although procaine hydrochloride is not generally considered to be a topical anesthetic King[5] indicates that surface (topical) applications of procaine hydrochloride will produce anesthesia of the mucous membranes. Our findings indicate that it should be particularly suitable as a local anesthetic for middle ear surgery. Further experiments are in progress to evaluate other currently used local and topical anesthetics.

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REFERENCES

1. Adriani, John: The Chemistry of Anesthesia. Springfield, Illinois, Charles C. Thomas, publisher, 1946.

2. Fowler, E. P. Jr., and Forbes, T. W.: Effect of Certain Agents on Cochlear Effect and Hearing. Proc. Soc. Exper. Biol. 32:827-829, 1935.

3. Fowler, E. P. Jr., and Forbes, T. W.: End Organ Deafness in Dogs Due to the Application of Certain Chemicals to the Round Window Membrane. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 45:859-864, 1936.

4. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics. New York, The Macmillan Company, 1958.

5. King, J. J.: Local Anesthesia in Otolaryngology and Rhinology. New York, Paul B. Hoeber, Inc., 1926.

6. Pitkin, G. P.: Conduction Anesthesia. Philadelphia, Pa., J. B. Lippincott, 1953.

7. Rahm, W. E., Strother, W. F., Gulick, W. L., and Crump, J. F.: The Effects of Topical Anesthetics upon the Ear. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 68:1037-1947, 1960.

8. Wever, E. G., and Bray, C. W.: The Effects of Chemical Substances upon the Electrical Responses of the Cochlea. I. The Application of Sodium Chloride to the Round Window Membrane. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:291-302, 1937.

LXXI

OTOLARYNGOLOGY AND COMPREHENSIVE CARE: A GLANCE INTO THE FUTURE

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There are certain problems confronting medicine, our specialty, and our hospitals which must be faced realistically and solved if we are to preserve our form of medical practice and our voluntary hospital system. These are problems which have resulted from an increasing public interest in health matters, with consequent demands for high standards of medical care, and an adequate distribution of this care, with a resulting expansion of hospital facilities, complicated by an inevitable increase in costs, and a steadily diminishing supply of professional personnel to meet these demands.

During the past 40 years we have seen tremendous developments in all fields of medical practice, as a result of great scientific advances, largely in biochemistry and physics. Never were there as many facilities or more funds available for research, aimed at the prevention and cure of disease, and the lengthening of life's span. We have only to look at our own specialty of otolaryngology to recognize the extent of this development. Indeed our field, like other areas of practice, has become so broadened that today rarely is one person capable of mastering all of its procedures and of being equally skilled in each of them. So we are beginning to see a further division into what might be termed sub-specialties, or at least areas of special interest. In addition we have seen the increasing use of paramedical skills; the audiologist, the clinical psychologist, the biochemist, the bacteriologist, the virologist, in addition to the medical social worker, vocational counselor, health educator, physical and occupational therapists; all contributing needed services.

In this period we have witnessed great expansion of hospital facilities. Hospitals, in the aggregate, now are considered our fifth

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Presented at the Tenth Annual Postgraduate Teaching Day in Otolaryngology, State University of New York, Upstate Medical Center, Syracuse, N. Y., April 21, 1960, and at the Annual Meeting of the Massachusetts Eye and Ear Infirmary Alumni Association, Boston, Mass., April 26, 1960.

largest industry; with over one and a half million patient beds, caring for over 25 million patients annually, with an average of two and a half employees per patient. And just as the hospitals have grown in number, so have their objectives been broadened to include education, research, and rehabilitation, in addition to care of the sick and injured. Great impetus to this expansion program was given by the passage of the Hospital Survey and Construction Act of 1946, the Hill-Burton program, which provides federal funds on a matching basis, and which has resulted in the development of needed hospital facilities throughout our country.

During this period there has been an awakening of the public conscience in the welfare of its people. To the old, inalienable "right to life, liberty and the pursuit of happiness" has been added the "right to health." Aside from the altruistic view-point, it became generally recognized that a healthy citizenry was an economic asset, and that it paid to return people to gainful employment, rather than to support them at the public purse; and that good medical and hospital care shortened morbidity and decreased mortality.

The public has become better informed on matters of health than ever before. Today there are over 200 full-time medical writers who provide newspapers and periodicals with coverage of all major medical and hospital meetings, as well as stories of new developments in medicine. By far the majority of these writers are able, conscientious, and qualified persons who are doing their jobs well. To be sure there are a few sensationalists who delight in destructive criticism, but these are in the distinct minority.

The public demand is for the best possible medical and hospital care, and it is a demand that must be met. We, physicians and our confreres in the health field, should recognize this and, working together, solve the problems that are involved.

I have been made especially cognizant of these problems as a result of experience as medical director of a hospital for over 25 years; as well as by service as Chairman of our State Advisory Committee on Health and Welfare, and of our State Hospital Advisory Commission since its inception in 1947. Perhaps, too, I am more aware of these because of practicing in the State of Maine. In 1956, under a grant from the Bingham Associates, and the Rockefeller Foundation, Dr. Wilson Smillie, Emeritus Professor of Preventive Medicine, Cornell University School of Medicine, conducted an exhaustive survey on "The Unmet Medical Needs of Maine." Among

other things this brought out that chronic illness and the disabilities of the aging were the primary health problems in Maine, and that the largest segments of our population were under 15 and over 65 years of age; neither group, with some exceptions being productive or self-supporting. From his studies, Dr. Smillie further concluded that this would be the pattern for the entire country by 1990. If this be so, perhaps our "Glance into the Future" may not be amiss.

With medicine possessed of these greater skills, with a rapidly expanding system of hospital facilities and a public better informed in matters of health and insistent upon these skills and facilities being available, what may be our major problems in meeting this demand?

I would list as most important the following:

1. Personnel—medical and paramedical.
2. Financing, including third party payments.
3. Care of the indigent and the geriatric patient.
4. Public relations.
5. Maintenance of standards.

PERSONNEL

Under personnel I include physicians, dentists, hospital administrators, nurses, technologists, and technicians, medical record librarians, social workers, physio- and occupational therapists, clinical psychologists, health educators, dieticians, biochemists, physicists, audiologists, special educationalists, podiatrists, vocational counselors, all those who work on the hospital team. Are there enough people with sufficient motivation in the numbers needed, to provide patient care of the quality and in the quantity demanded?

Perhaps of most concern is the diminishing supply of physicians in all areas of practice. We, in otolaryngology, have been disturbed by the smaller numbers of medical graduates electing our field and we have even had our prophets of gloom, saying that ours was a dying specialty. Not so, unless medicine itself is dying. The U. S. Public Health Service states that the number of graduates from medical schools of this country this past year (7,400) was 3,600 short of the number needed to keep the proper ratio of our population, which is increasing year by year. In 1959 the number of graduates of our liberal arts colleges entering medical schools was nine per cent less

than the previous year, and that year's number was alarmingly low. Consider how many of our interneships and residencies now are filled by graduates from foreign countries. Our medical school quotas are not by any means filled and some schools no longer are as critical of the college grades of their applicants as formerly.

In asking college students if they are considering medicine as a career I have met too frequently with, "It's too hard, too long, and I can make more money doing something else." And then, "It's two extra years in the Army."

Oh! certain of the old established schools have had no trouble, at least as yet, and seem altogether too complacent. There sometimes has been a tendency for the ivory tower to be complacent, unconcerned about the rising tide, until, too late, it finds itself engulfed. Isn't it time that some of our medical educators came down from the ivory tower, and with the feet, perhaps, a little bit wet from the rising tide, begin to seek a solution to this problem? It is indeed refreshing to note the experiments at Johns Hopkins, Western Reserve, and Boston University.

I am somewhat sympathetic with the college senior when he stated "it was too long." A minimum of 15 years to qualify for Board examination in our specialties is a long time. And modern medicine, if it is to make proper use of all of its wonderful advances, needs specialists, well-qualified specialists. I think we might take a second look at some of our residency programs to see if some of the fat could not be eliminated. The practice of medicine demands continuing programs of education throughout one's career, for medicine is not static. Each day there are new things to learn, and last year's residency program is soon out-dated by new developments. If careful appraisal could suggest means to speed up the medical school and residency programs, without sacrifice of any essentials, it would certainly seem worthy of consideration.

It may be interesting, if not consoling, to note that both the ministry and education are faced with the same problem. I had known that fewer able and dedicated young people were seeking careers in teaching but I was surprised to learn first-hand from Catholic priests, Protestant ministers, and Jewish rabbis that all were facing similar situations.

It seems to be a matter of proper motivation, with need for greater emphasis upon idealism, rather than upon materialism. This

must start before medical school, in college, or even better, in high school. Dr. Conant's criticism of the latter merits careful study. Perhaps the answer is to develop better, more dedicated teachers, more interested in guidance, in our secondary schools.

For the prize example of a confused program one has only to look at nursing education today, with two year, three year, four year, and five year programs, and with bedside teaching often relegated into a secondary place in the background. Here we seem to have entrenched bureaucracy with a strangle-hold on the whole profession, resembling some of our most militant labor unions. Add to that, the tremendous attrition due to marriage and its sequelae, and it is not hard to visualize the difficulty. Honorable Frances Bolton, Congresswoman from Ohio, made a survey of the time actually spent in nursing by all of the graduates of our nursing schools for a five year period, and found it averaged nine months. We have seen the development of programs for practical nurses, nurses' aides, and surgical technical aides, with the increasing use of team nursing under a registered nurse; which, incidentally, seems to function quite satisfactorily. The greatest deficiency in nursing today is at the administrative and supervisory level and this, indeed, is serious. Undoubtedly the natural urge to marry and raise a family deters the majority of these young women from any desire for professional advancement. It may be that in the future we shall see nursing largely a male profession, at least in the higher grades. As the man generally is the breadwinner of the family, this might have the advantage of stability. Rapid turn-over of personnel is expensive and impairs efficiency. To a certain degree the same holds true of other paramedical fields and we are seeing a greater number of male technicians, therapists, social workers, and even hospital housekeepers.

FINANCING

Financing of any program of patient care is a serious problem, in which politics often rears its ugly head, but one which eventually must be settled by the public who have to pay the bills, either personally, through the purchase of insurance, individual or group, or by taxation. All of these methods, in the final analysis, take it out of the same pocket, yours and mine and our neighbors. And it is a "good buy" only if equitably borne and it results in adequate service.

Interestingly enough, well-conducted surveys have shown that the greatest resentment is directed against hospital costs and that there is relatively lesser complaint against doctors' fees. This phase of our

economy has been effected, like all others, by the prevailing and apparently ever-increasing inflation. Of course, hospital costs are high. Today's standards of patient-care call for increased numbers of skilled personnel, expanded equipment, expensive procedures, and costly drugs.

The fact that this has resulted in lowered mortality and shortened morbidity often is overlooked. Unfortunately the average person, too often has an erroneous conception of what is involved. He doesn't understand, perhaps doesn't wish to, for he is paying for something he really doesn't want. He doesn't want to be sick, resents illness as a threat to his individuality, to his adulthood.

By far the largest part of the hospital dollar is expended for the salaries and wages of the many people required to operate the institution. For many years hospital salaries and wages have not been comparable to those in Industry. Often they were so low in certain categories as to attract only submarginal workers. This is being corrected, and must be, for we need qualified personnel if we are to provide the necessary standards of care. The threat of unionization, with all it entails, cannot be ignored.

Increasing use of insurance coverage, Blue Cross, Blue Shield, Industrial and Union plans, as well as private policies, has done a great deal towards maintaining solvency, both to patient and hospitals. In our own institution 67 per cent of hospital accounts last year were covered by some form of insurance. The abuse of coverage, however, may threaten the solvency of the plans and result in an inevitable increase in premiums, causing discontent, if not rebellion, on the part of the purchaser. At the present time we are conducting a utilization study to provide information aimed at correction of this problem. A special form is attached to every patient's record on which the attending physician notes the cause for admission, the predicted length of stay and checks off any possible services which might have made admission or long stay avoidable. In this connection it is essential that members of our profession realize their responsibilities in safeguarding the solvency of these plans. There have been regrettable instances of padding and overcharging upon the part of certain unscrupulous physicians which reflects upon the whole profession.

CARE OF THE INDIGENT AND GERIATRIC PATIENT

The problem of care of the indigent and the geriatric patients has two components: availability of beds and financing the cost of

care. A means test cannot be a standard for hospital admission of the medically indigent and as "we have added years to Life, it is also our responsibility to add Life to years."

These cases tend to a comparatively long hospital occupancy. A survey, conducted at the Thayer Hospital, showed that the average stay of private patients was 5.7 days, while that of the indigent only partially covered by governmental sources was 13.1. While certain of these cases were more serious, often due to previous neglect, in many the reason for the longer stay was either the reluctance of the patient to return home, the unwillingness of his family to take care of him, or to lack of suitable home conditions, or distance from the hospital. In some cases the attending physician, finding it easier to care for the patient in the hospital, even when distance was no problem, has failed to make proper use of out-patient facilities, thus contributing to improper utilization of beds and added costs to the hospital. Bearing in mind the increasing number of geriatric and long-term chronic cases, more intelligent planning in future construction would seem economically sound. What might be considered as nursing home facilities to provide convalescent care in conjunction with, and operated by the hospital on a non-profit basis, could be constructed at a cost of from one-fourth to one-third of general hospital construction and would provide satisfactory care, something often lacking in the proprietary nursing home.

In states such as mine which provide inadequate payment for the care of indigent patients, there is an added problem. With an average cost of over $28.00 per day and reimbursement from governmental sources ranging from less than seven to $12.00 per day, depending upon the categories, the hospital faces a serious loss.

The inevitable deficit due to caring for the indigent can be met only by increasing the charges to private or paying patients, unless there be adequate endowment income or sufficient contributions from community chests, foundations, or private charitable sources. Our changing economy has resulted in a shrinking of many private fortunes and the foundations have so many demands made upon them that contributions from these sources rarely are sufficient to meet current losses. The increased charges to the private patient actually means a special tax to help defray the cost of caring for the indigent, a tax imposed upon a person already handicapped by illness and prevented from gainful employment. Certainly this would seem to be a justifiable cause for complaint. C. Rufus Rorem, Ph.D., executive director, Hospital Planning Association of Allegheny County, Pa.,

recently suggested that this amount might be designated as a "surcharge for charity" on the patient's bill, in order that he could use it as an income tax deduction. Of course, the real solution lies in arousing public opinion sufficiently to demand that the cost of care of the indigent be met from public governmental funds, an already accomplished act in certain states.

Canada recently adopted a plan for universal coverage, regardless of economic status, without limitation of time for hospital stay, at a cost of $4.20 per month per family, or $2.10 per month for single persons. For years Maine and Canada have enjoyed reciprocal hospital relations in our border towns. Patients and physicians used hospitals on either side of the boundary. Now the Canadian hospitals are so filled with needless and long-term admissions that our own border communities are in dire straits and we are faced with the problem of undertaking a large amount of new hospital construction in that area. This may give us some idea of what might well be the effect of the Forand bill now before Congress. And now, labor is demanding a plan similar to Canada's, "financed jointly by employees, employers, and the government, for the complete medical and hospital care of every man, woman, and child in America." Laudable as this may sound to many, just consider it realistically for a moment. Obviously it implies government medicine and hospitals, with bureaucratic control as to admissions. With its inevitable over-utilization and lengthened stay, how could the critically ill, acute cases be assured of needed admission? Would the public really approve of this if it understood what it implies? Admissions should be based upon medical need which can be measured only by the physician, not by a political bureaucrat.

Naturally one might question if this is any concern of otolaryngology. It becomes so only as it concerns medicine as a whole, as a profession that is dependent upon the solvency of our hospitals. It seems unfortunate that it threatens to become a matter to be dealt with politically, and probably unwisely, unless we interest ourselves in developing a better understanding of these problems on the part of the general public. This is largely a matter of public education.

PUBLIC RELATIONS

Lest we become complacent as a result of the comparatively lesser criticism of professional fees, there is definite evidence of an increasing discontent with the degree of availability of medical care and a more critical appraisal of its quality. The former hallowed dignity of the

physician has disappeared. No longer is the doctor the revered, sainted person of our grandfathers' day. Too often now, he has assumed the character of an impersonal, objective, though talented, technician.

Periods such as these, in which we are living, are favorable ones for controversies, destructive criticisms, and political demagoguery. While the medical press is doing an excellent job, its good effects may be penalized by individual acts or unhappy personal experiences. After all, public relations are simply personal relations in the aggregate.

The shortage of physicians, the increasing demands for medical services, with added burdens upon the doctor, and, in some instances, perhaps an urge for increased financial returns, have a tendency toward what may be termed "production line practice." This is not the best type of medicine. It might be termed "target medicine" and as such is open to justifiable criticism. No specialty, including ours, is immune to this.

MAINTENANCE OF STANDARDS

Our greatest responsibility as physicians is in developing and maintaining the highest possible standards of patient care, for a public better informed and more critical than ever before. While more easily achieved in the hospital through the controls incident to accreditation, these standards are just as necessary in office practice. For it is in the latter that often the error is initiated, which subsequently may be compounded in the hospital. The temptation to do "target medicine" may be difficult to forego when confronted with a busy schedule. Yet it should not be condoned.

Since adding a department of rehabilitation in our hospital I have been greatly impressed with its effect, perhaps psychological, upon the entire program of patient care. Rehabilitation has become a glamorous word, often considered as something new, and too frequently, as synonymous with physical medicine. Actually rehabilitation means the "restoration to former capacity," in our profession to "health." In clinical practice this can be construed as comprehensive care, aimed at achieving the optimum in convalescence. Should not this be our goal? Comprehensive care implies a broad conception of the problem, utilizing all indicated skills, both medical and paramedical. Too often the clinician fails to see beyond his own particular field. Mosher

once said, "The strength of specialism is specialism, and, paradoxically, the weakness of specialism is specialism."

McMahon in his presidential address to the American Laryngological Association in 1956 said, "the inclination has been more and more to consider the patient as a whole, one in whom otolaryngologic symptoms often may be considered a local manifestation of constitutional dyscrasias." Indeed the conscientious otolaryngologist rarely can do full justice to a patient with any serious difficulty without careful consideration of the whole man; nor is he doing his whole duty to his patient when he overcomes the immediate disease disability, but neglects using all means of restoring him to a useful and self-sufficient place in our economy. "It is wasteful to spend large sums on expensive hospital care, only to return a man to living and working conditions that are almost certain to drive him back to the hospital in a few months." [1]

Frequently findings in the ear, nose, and throat may be the early, perhaps the only, signs of a systemic or constitutional disease. The field of otolaryngology may be a veritable show-window of many acute and chronic diseases. The oral cavity may provide clues of many significant general conditions. The old-time physician, while lacking the advantages of modern laboratory tests, gained much valuable information by inspection of the tongue. A poor dental condition may be a real factor in a nutritional problem. The lips may suggest avitaminosis. Similarly the nose may reveal much more than a septal deviation or signs of sinusitis, while the naso-pharynx may present clues of great diagnostic value. Routine laryngeal examination may reveal evidence of an unsuspected thoracic lesion. Allergic states should be readily recognized in the nose but often may be overlooked in the patient with eczema of the external ear, otitis media with effusion, or a flareup of a previously dry chronic suppurative otitis media or radical mastoidectomy cavity.

Every case of vertigo may not be Ménière's disease and often medical and neurological consultation is more effective than empiric use of vasodilatation and blocking agents. Careful study may indicate, perhaps, a central condition, or, more fortunately, postural hypotension, hypometabolism, or even the hyperventilation syndrome as the cause.

Often we see patients with vague bizarre symptoms of scratchy irritable throats, dry nose, or of hoarseness in whom a carefully taken history, plus an overall look at their appearance, suggest subclinical

myxedema and the advisability of radio-active iodine or protein-bound serum iodine tests, rather than futile local applications.

Furunculosis of the nasal vestibule or of the external auditory meatus, especially if recurrent, should suggest the possibility of an underlying diabetes. Epistaxis, especially if recurring, should merit consultation with the internist. Often hypertension may be a pertinent factor, and recently we uncovered a case of DeGugliemo's disease in such a patient.

Comprehensive care often extends beyond the medical phase. Naturally when we see patients with irreversible hearing loss we advise auditory rehabilitation. But should we stop there? Should not we consider the possible environmental or emotional factors and make use of all available services to correct these? I have in mind a young man, 20 years of age, an epileptic, who has a very severe nerve deafness resulting from mumps labyrinthitis at the age of 15. While he obtains some slight benefit from a hearing aid on one ear, obviously he should have had auditory rehabilitation five years ago. Instead he had a tonsillectomy. His education had been sadly neglected. He was without employment or funds, and was emotionally disturbed. So, in addition to training in speech-reading and medical treatment of his epilepsy, vocational rehabilitation was provided to train him and place him in a job he can handle. He is now gainfully employed as a meat cutter. This, in itself, is good medicine.

We have a young girl with a very severe perceptive deafness resulting from meningitis at the age of two, who has been in our program of auditory rehabilitation. She is an excellent speech reader and is doing well, now in her senior year in high school. Recently she became emotionally disturbed because of worries about her future career. She has an intense desire to become a nurse which is impractical. When told we would train her, either as a tissue technician or a physiotherapist, she became quite contented and has continued her school program in a very satisfactory manner.

Another young girl had developed an oro-antral fistula, secondary to a dentigerous cyst. She was a most pathetic creature; shy, withdrawing from all social contacts, because of Hirschsprung's disease, megacolon, with its disagreeable bodily odors. Repair of the fistula was of secondary importance to a long program of patient training and education as to the management of her colon. Now she is training as a nurses' aide, with the eventual hope of entering a school of

practical nurses, and is already a changed individual. People desire to be useful, to have a place in our society.

A young man of 32 was referred because of a speech defect of six years' duration due to inability to pronounce labials. He had lost control of his lower lip which he had to close manually. Careful examination revealed lesser but similar trouble with the lower eyelids. It took a great deal of group study, including the collection of earlier pictures of himself, and of his deceased father, to arrive at a diagnosis of Landouzey-Dejerine dystrophy.

Each contact a patient makes with a physician is a golden opportunity that should not be lightly handled by "target medicine." Certainly the patient who appears with an acute otitis media, or a peritonsillar abscess demands acute, specific care. But with many patients, observation must transcend the organ and look for systemic disease; or for conditions that decrease the body reserves, such as nutritional or fatigue factors; or must see the symptom as evidence that the patient is unable to cope with his environment. This broadened role of the doctor is comprehensive care. To do proper comprehensive care the otolaryngologist should have a knowledge of the general diseases that can affect the organs of his specialty. He must recognize the indications for consultations and make effective use of them.

Comprehensive medicine may conflict with the theory of single causation. However, it has been proven that many causes must be considered, both in the prevention of recurrence and in rehabilitation. If we accept the theory of multiple causation, the physician's role necessarily must be broader and, to play this role effectively, he must work closely with his colleagues and with paramedical personnel. This calls for knowledge, experience, judgment, and thoroughness, together with a willingness to devote all necessary time to patients seeking help. It cannot be done on a production-line basis. And often it cannot be done alone. But, with the resources of the modern hospital and the special medical skills so readily available today, comprehensive care should present a challenge, not an impossibility to be disregarded. Restoration to the optimum is a goal worth striving for.

CONCLUSION

We are living in a changing world. Viewed in retrospect the past four decades have been a revolutionary period characterized by great scientific developments and by socio-economic and political changes which have had and will continue to have a tremendous impact upon

our profession, our hospitals, and the entire health program of our country. These have resulted in a public demand for a program of the best medical and hospital care, and adequate distribution of that care, to be paid for on an equitable basis.

This involves problems of supply of personnel, of financing, of care of the indigent and geriatric patient, of public relations, and of maintenance of high standards. Solution of these problems requires the co-operation of all working in the health field. Development and maintenance of high standards is a particular responsibility of medicine. The concept of comprehensive care implies the highest standards and leads to the desired goal. As members of an important and necessary specialty of medicine this should be a concern of every otolaryngologist.

DISEASE

Acute, Incidental Disease, such as otitis media, sinusitis

Need Acute Care Only

Subacute, Recurring Disease, such as Meniere's, nasal polyposis

Need Care, Plus Thorough Evaluation

Chronic Disease, such as malignancy, chronic suppurative otitis media, deafness

Need Care, Evaluation, and Rehabilitation

Bizarre Type, such as positional vertigo, functional aphonia

Need Evaluation and Treatment

Palliative Type, such as terminal cancer

Need Palliation Plus Respect

Thayer Hospital

REFERENCE

1. Ferguson and MacPhail: Hospital and Community. Oxford Un. Press, 1954.

LXXII

MELKERSON'S SYNDROME

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Melkerson's syndrome is a rare, peculiar entity which has been adequately described in foreign literature (mainly Scandinavian) but is quite unknown in other parts of the world.

Melkerson described the syndrome in 1928. Since 1950, according to the Current List of Medical Literature, 34 articles dealing with the subject were published. Of these only five are in English.[1-5]

The syndrome comprises the following triad of symptoms:

1. Peripheral facial palsy, which may be familial, often bilateral and which may show a tendency to relapse;

2. Angioneurotic facial edema, especially of the lips, and

3. Lingua plicata.

The paucity of such reports in English literature, the lack of reports on adequate biopsies taken from the lips, cheek and tongue and the results of treating this condition with the corticosteroids has prompted me to report such a case which was recently observed at the American University Hospital.

REPORT OF A CASE

S.K., case number 145180, a 25 year old woman, suffered from left-sided facial paralysis at the age of 16. The paralysis regressed after two months and she was well until the age of 22 when she had another attack of facial paralysis, this time on the right side. This also regressed after a few months and her appearance returned to normal. At the age of 24 her upper lip swelled suddenly but subsided entirely within ten days. Since then she has been getting these recur-

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Fig. 1.—Appearance of face during an attack of swelling.

rent swellings every six to eight weeks and during the past year the swellings have extended to include the left parotid area, both lips and the tongue.

Physical examination on admission revealed no residue of facial paralysis. Her upper lip and the left side of the face were slightly swollen (Fig. 1). The swelling was not tender, not pitting and there was no discoloration of the skin. The mouth, teeth, nose, nasopharynx and throat were normal. The tongue was markedly furrowed (plicated) (Fig. 2). The rest of the physical examination was negative.

Laboratory data revealed the following: Hemoglobin 11.18 gms, W.B.C. 10400, neutrophils 54, Band 8, Lymphocytes 30, Monocytes 4, Eosinophils 6. BUN 16 mgs%. Proteins 7.9 gms%, Albumen 4.8 gms%, Globulin 3.1 gms%, Blood Sugar 78 mgs%. Cholesterol 165 mgs%. Urinalysis was negative. VDRL and Kolmer were nonreactive.

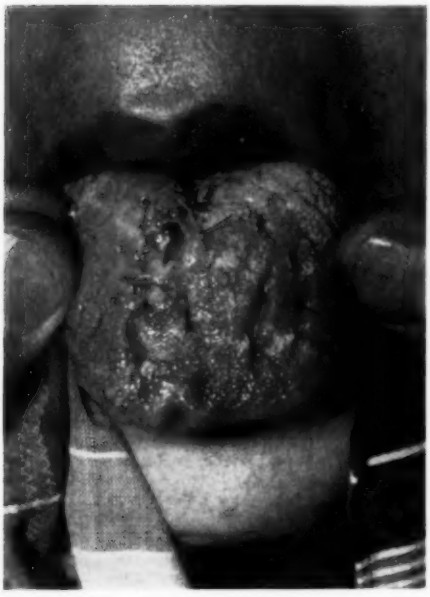

Fig. 2.—Plicated tongue.

X-rays of the sinuses, lower jaw, teeth and chest were normal. Audiograms were normal. Skin tests for the various common allergens were all negative.

The results of lumbar puncture were unremarkable.

Several biopsies were obtained from the dorsum of the tongue and from the upper lip and cheek. The pathologist's report was as follows: "Sections of the tongue biopsy demonstrate irregular thickening of the stratified squamous epithelium with formation of hyperplastic rete pegs dipping into the subepithelial stroma (Fig. 3). The latter shows considerable fibrosis with dilatation of endothelium-lined channels (probably lymphatic vessels) together with perivascular infiltration by lymphocytes, histiocytes and plasma cells (Figs. 4 and 5). The fibrosis, vascular dilatation and round cell infiltration extend deep into the muscle layer.

"Sections of the buccal mucosa and cheek show essentially the same findings mentioned above. The inflammatory reaction is more

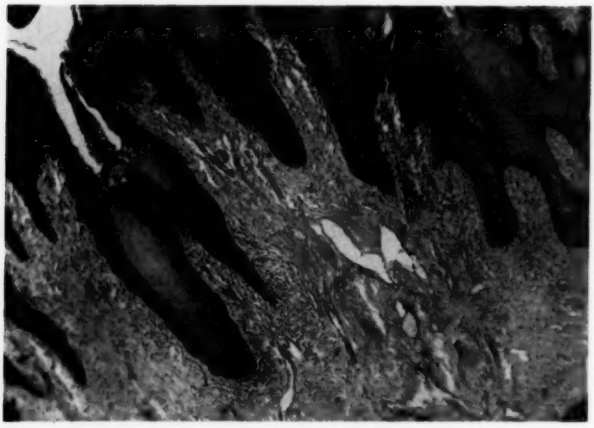

Fig. 3 (X 68).—Biopsy of tongue with severe lingua plicata. There is irregular thickening and hyperplasia of the squamous epithelium. The subepithelial stroma shows lymphatic dilatation, fibrosis, and chronic inflammation.

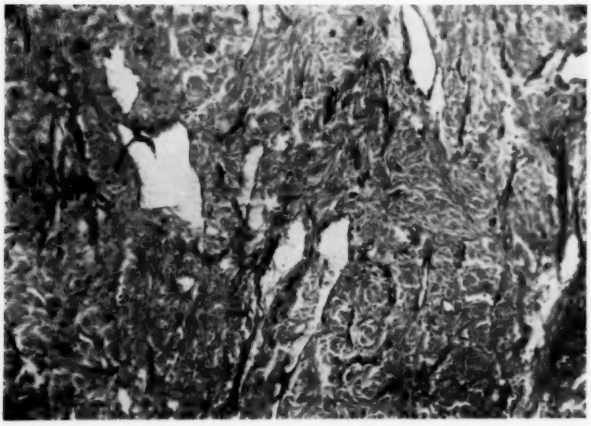

Fig. 4 (X 83).—Note the lymphatic dilatation, fibrosis and lymphocytic reaction in the subepithelial stroma of the tongue.

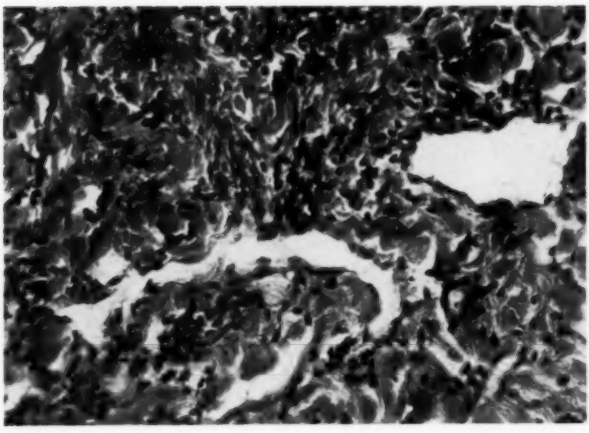

Fig. 5 (X 500).—Tongue. Note the dense lymphocytic perivascular infiltration around the small endothelium-lined channels.

pronounced and is seen predominantly around blood capillaries and arterioles. Some lymph follicles are formed (Fig. 6) and some of the lymphocytic aggregates contain histiocytes and epithelioid cells giving the appearance of a granulomatous reaction. A few eosinophils are encountered. In addition, there is marked interstitial edema with widening of the tissue spaces between muscle fibres."

It was decided to try the effect of cortisone administration on the lesion but the swelling subsided a few days after the patient was admitted to hospital. She was therefore discharged with instructions to report as soon as a fresh swelling appeared. This she did and on December 15, 1959, she was readmitted with an extensive swelling including the upper lip, left side of the face and tongue. She was started on medrol (methylprednisolone, 4 mg tablets), four tablets daily. At the end of two weeks the swelling had subsided markedly but the patient stated that the medication did not hasten or alter the course of the attack in any way. In fact, this time the attack lasted a little longer than usual. Nevertheless, medrol was continued indefinitely to find out whether or not an attack would occur under its cover. After taking the medication for five weeks she came down with another attack of facial swelling similar in all respects to the previous ones. This greatly dampened our enthusiasm and therapy was discontinued.

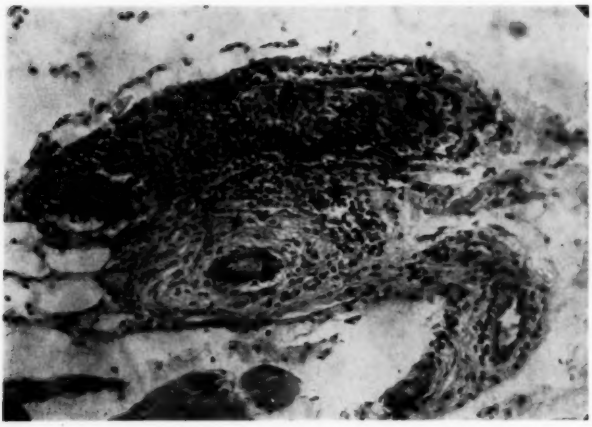

Fig. 6 (X 218).—Cheek. Perivascular inflammatory reaction. Note the lymph follicle next to a focus of perivasculitis around an arteriole.

COMMENT

This case is strikingly similar to other cases reported by various authors. Peripheral facial palsy began in youth. Many years elapsed before swelling of the face appeared, although in some cases the two symptoms occur simultaneously. At first the swellings subsided completely after each attack but later on there was a permanent residual swelling of the upper lip and left cheek. Swelling of the floor of the mouth and tongue which occurred in this case has been reported by other authors. The skin over the affected parts appeared natural, did not pit or show other inflammatory signs or regional adenitis.

As in other cases the furrowed tongue in this case was present since childhood, a long time before the other symptoms appeared. None of her immediate family showed this condition. It is unknown why facial palsy and facial edema develop in patients with lingua plicata. Ekbom[1] has shown that the incidence of plicated tongue was about four times greater in patients who develop cryptogenic facial palsy as compared with a control series of normal individuals.

Study of microscopic sections excised from the edematous facial areas did not differ in any way from previously reported cases. Interestingly, the pathologic anatomy of the tongue, which has not hitherto been reported, showed a similar picture. Dr. Azar, pathologist at

the University, has the following comments to make on the case: "The pathogenesis of Melkerson's syndrome has given rise to considerable speculation. According to Kettel[3] the facial edema is a form of angioneurotic edema caused by a disturbance in the regulation of vasomotor fibers in the facial nerve. McGovern[2] seems to believe that the facial swelling is the result of a chronic inflammatory process of the corium of the cheek resulting in a secondary obstruction of lymphatic channels.

"In this case we have been tempted to give primary significance to the perivasculitis which is observed in both the tongue and cheek biopsies. The lymphatic dilatation may well be, as suggested by McGovern, secondary to chronic inflammation and fibrosis. Irritation of the facial nerve may also be the result of edema, chronic inflammation and fibrosis. It is reasonable to assume that the essential process in Melkerson's syndrome is that of an allergic vasculitis not unlike that of serum sickness and some of the so-called collagen diseases such as periarteritis nodosa. However, the vascular involvement in this syndrome is limited to the tongue and cheek in contradistinction to the generalized distribution of the vascular lesions in 'collagen' diseases. This focal rather than diffuse involvement of the vascular process may be attributed to the type of allergen, its mode of entry as well as some form of locus minoris resistentiae which contribute to make the tongue, mouth and cheek the target organs in this syndrome."

Put in simple language, our pathologists' comment merely states that, so far, nothing is known about the etiology or pathogenesis of Melkerson's syndrome. Whether or not it is justifiable to place this entity within the group of collagen diseases remains to be seen. Micropathologic appearance of the lesions seems insufficient to warrant such a hypothesis; and if the lack of response to corticosteroid therapy is a valid criterion, it makes the hypothesis even less likely.

Aside from such antiquated procedures as injection of boiling water into or excision of swollen parts treatment of this condition may be said not to exist. Kettel[4] states that cortisone administration has proved useful for the swollen lips but that neither this treatment nor partial excision of the lips prevents a recurrence of the swelling. There was no response to cortisone therapy in the case under discussion. Decompression of the facial nerve when facial paralysis does not recover spontaneously after a reasonable length of time, as suggested by Kettel, may be tried. The opportunity hardly arises, however.

SUMMARY

Melkerson's syndrome consists of recurrent paralysis of the facial nerve, edema of the face and lingua plicata.

The etiology of the disease is unknown. Further evidence is needed to classify it with the collagen diseases.

Treatment of one case with cortisone failed to hasten recovery or prevent recurrence of the facial edema.

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I am grateful to Mrs. Joan Chippindall for her assistance in preparing this paper.

REFERENCES

1. Ekbom, K. A.: Plicated Tongue in Melkerson's Syndrome and in Paralysis of the Facial Nerve. Acta Medica Scandinavia 138:42, 1950.

2. McGovern, F. H.: Melkerson's Syndrome. Arch. of Otolaryngol. 51:371, 1950.

3. Auckland, G.: Melkerson's Syndrome. Brit. Jour. of Dermatol. 70:458, 1958.

4. Kettel, K.: Surgical Treatment in Atraumatic Facial Paralysis. Jour. of Laryngol. and Otol. 73:491, 1959.

5. Kettel, K.: Melkerson's Syndrome. Arch. of Otolaryngol. 46:341, 1947.

LXXIII

THE EFFECTS OF ABNORMAL BODY TEMPERATURE UPON THE EAR: HEATING

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Newark, Del.

Data from an earlier paper[2] indicate that reductions in body temperature are accompanied by reductions in the magnitude of the cochlear response. In order that these data might be more fully understood, it became apparent that the effects of increased body temperature upon the response should be studied.

While studying the decline in the cochlear response after death, Wever, Bray, and Lawrence[4] noticed that there occurred a slight increase in the response when a lamp located above the animal was turned on. The effect, though slight, was consistent through three trials. At one point during the same experiment a cautery was inserted in the opening of the bulla and brought near the cochlea. This action resulted in a rapid rise in the response (about four db) followed by a serious fall.

These observations concerned the effect of cochlear temperature on the magnitude of the cochlear response. Up to the present there has been no study of the relationship between this response and hyperthermic states. The purpose of the present experiment was to investigate this relationship. Recovery from temporary hyperthermia was included in this study.

METHOD

Anesthetized guinea pigs were used as experimental animals. A 1000 cps signal produced by an audio-oscillator (Hewlett Packard,

From the Physiology Laboratory, Department of Psychology, University of Delaware.

200 AB) was fed into a loudspeaker. Aerial sound was conducted into the external meatus through a tube leading from the loudspeaker. The resulting cochlear potentials picked up at the round window by a platinum foil electrode were measured with an audio frequency spectrometer (Brüel and Kjaer, 2109) operating as a selective voltmeter. The preparations were isolated within an electrically shielded room during recording procedures.

The body temperature of the guinea pig was measured with a laboratory thermometer placed under the side of the animal as he lay on the operating table. The suitability of this method of measuring body temperature is discussed elsewhere.[2]

Air temperature at the bulla, measured with a second thermometer, was held constant at 26° C. ± 1°. Thus, any substantial change in the cochlear response which accompanied changes in body temperature was assumed not to be due to significant temperature changes within the cochlea itself.

Raising body temperature above normal was effected with a reflecting lamp located approximately 60 cm above the animal. The heat emitted from this lamp was sufficient to produce a slow increase in the animal's body temperature to the desired level. The skin area which was warmed extended from the lower thoracic region in a posterior direction and included the hind quarters. The entire head region and the bulla thermometer were shielded from the heat source.

The return of body temperature to normal was effected by the removal of the heat source and the application of a cool water pack (approximately 20° C). The cool pack, supported by steel rods, was held in close proximity to the animal.

Severe hyperthermia was produced in four animals until they expired. Seven animals were used to investigate whether or not the effects of heating were reversible, but because of the guinea pig's high susceptibility to hyperthermic death, four animals expired before body temperature returned to normal.

RESULTS

The data in Figure 1 show that conditions of artificially induced hyperthermia produce a loss in the magnitude of the cochlear response. The decline in the response was similar among the animals. For the temperature change from 37° to 40° C. slight losses occurred (1 to 2

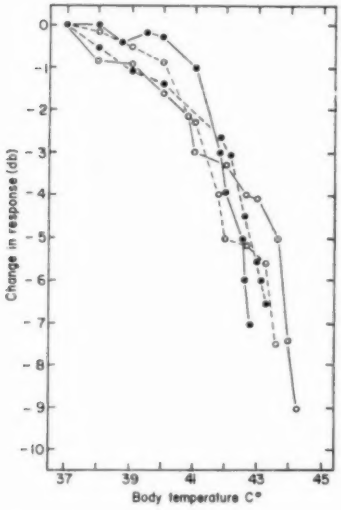

Fig. 1.—The effects of increased body temperature upon the magnitude of the cochlear response to a 1000 cps tone for each of four guinea pigs. The changes in response are given in decibels relative to the output at normal body temperature (37° C.). Rates of heating approximated 6 degrees C. per hour.

db). However, further increases in body temperature produced more marked losses, one as extensive as 9 db. The rate of temperature change was essentially the same for the four animals (6° C. per hour). Whether or not the drop in response is in some way related to the rate at which body temperature changes remains to be demonstrated.

Data gathered on three animals to investigate recovery are presented in Figures 2, 3, and 4. As shown in Figure 2, the animal's response dropped 4 db when body temperature was raised to 41.2° C. No recovery occurred when body temperature was returned to normal. Instead, the cochlear potential dropped another decibel within the next 90 minutes. Thereafter it remained constant until the animal expired.

Recovery data from the second animal are presented in Figure 3. The procedure was identical to that followed for the previous animal, except that the rate of temperature change was slightly slower. When

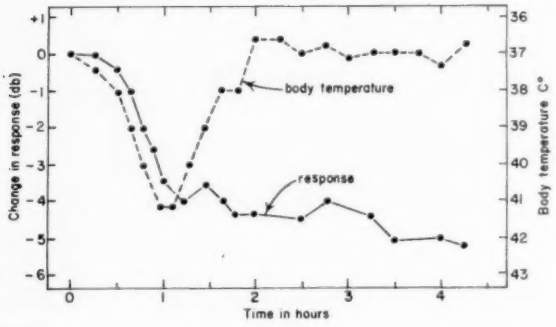

Fig. 2.—Curves showing the relationship between body temperature and the loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

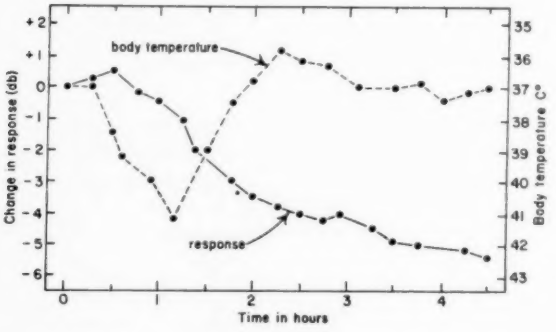

Fig. 3.—Curves showing the relationship between body temperature and the loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

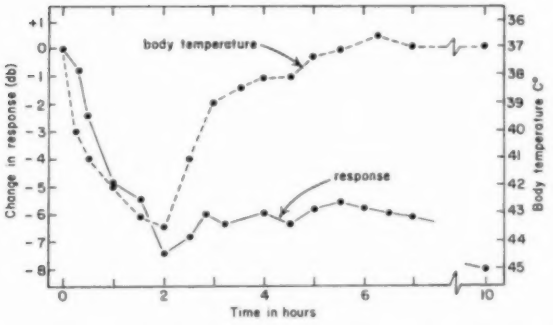

Fig. 4.—Curves showing the relationship between body temperature and the loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

body temperature reached 41.2° C, the loss in the response amounted to 1 db. While temperature was returned to normal, the response continued to decline until the loss amounted to 5 db. Thereafter, no further loss was noted until the animal expired.

The third animal on which recovery was investigated underwent the most severe temperature changes. Two hours after heat was applied, body temperature had risen to 43.5° C. During this time the response suffered a loss of about 7.5 db. Restoration of normal body temperature produced partial recovery (1.5 db), but at the time of death the response showed an 8 db loss. These data are presented in Figure 4.

COMMENT

The data from the present experiment indicate that there exists an inverse relationship between the severity of hyperthermia and the magnitude of the cochlear response. As the temperature of the body increases above its normal level, the cochlear response decreases at a rate which is related to the extent of the temperature deviation. The decrease in response observed during hyperthermia is very similar to that noted in our earlier study of hypothermia. For a comparison of the relationships between body temperature deviations and changes in the magnitude of the cochlear response, composite functions are pre-

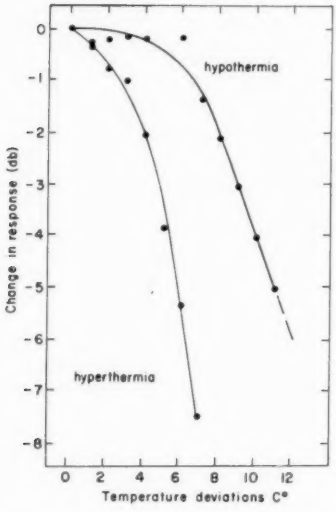

Fig. 5.—Composite functions showing the relationship between body temperature deviations and changes in the magnitude of the cochlear response (in decibels relative to the output at normal body temperature). Body temperature is given in deviations from normal (37° C.). Each curve is based upon observations made on each of four guinea pigs. Hypothermic data from Gulick and Cutt, 1960.[2]

sented in Figure 5 which show the losses (db) obtained during both hypo- and hyperthermia. Each curve is based upon observations made on four guinea pigs. Further similarities in the effects of these two conditions may be noted in Figure 6 wherein intensity functions taken at extreme temperatures are compared with the normal intensity function.

Besides the similarities in the effects of heating and cooling upon the cochlear response, three important differences may be noted. First, a more consistent pattern of response change occurred as a result of rising temperatures than occurred as a result of decreasing temperatures. Second, some reductions in the cochlear potential incurred during hypothermia were fully reversed by the restoration of normal body temperature. Only partial recovery (1 db) was observed following hyperthermia and in only one animal. Third, data suggest that the guinea pig is more susceptible to a hyper- than to a hypothermic death. Death in both conditions resulted apparently from

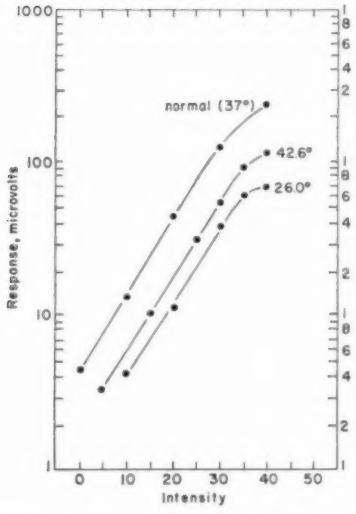

Fig. 6.—Intensity functions plotted for a 1000 cps tone at 37° (normal), 26°, and 42.6° C. Intensity values are expressed in decibels relative to .004 volts output at the oscillator.

respiratory failure. Other evidence supports this observation but suggests that circulatory inadequacy might precede respiratory failure.[1,3]

The most obvious way for abnormal body temperature to affect the magnitude of the cochlear response would be for the temperature change to influence directly the hair cells of the inner ear and thereby reduce their output. This interpretation, however, seems unjustified because of the likelihood that the cochlear temperature remained constant. Three lines of reasoning support the inference that cochlear temperature did not fluctuate appreciably. First, the head region generally underwent less severe temperature changes than the peritoneal temperatures would indicate because the head of the animal was shielded from both the hot and the cold stimuli applied to the body. Second, the warm blood circulated toward the ear would tend to be cooled by the tissues in the head region through which the blood must circulate. Moreover, because only a small amount of blood circulates through the stria vascularis there would be little opportunity for an exchange of heat to occur between the endolymph bounding the hair cells and the blood in the stria vascularis. Third, the cochlear

bone is extremely thin so that laboratory air (26° C. ± 1) circulating within the middle ear would also tend to maintain the cochlea near room temperature.

If we assume that, for the reasons indicated, the cochlear temperature did not vary significantly, then we must look somewhere other than the cochlea itself for an explanation of the losses noted during hypo- and hyperthermia. At the moment it seems reasonable to suggest that the temperature changes were altering one or more of the mediating processes which contribute to the generation of the cochlear response.

It should be pointed out that the hair cells of the inner ear are probably operating at their maximum efficiency under conditions of normal body temperature since temperature deviations in either direction result in a loss in the magnitude of the response. Nevertheless, in the guinea pig the range of body temperature within which the response seems to remain relatively constant is rather extensive (32 to 40° C.).

Further experiments are in progress to ascertain the nature of the effects of temperature upon the cochlear output.

SUMMARY

An increase in the body temperature of the guinea pig causes a reduction in the electrical response of the cochlea. The effects of hyperthermia follow closely those already reported for hypothermia. The body temperature of four animals was raised until each expired due to hyperthermia, and three others were used to study recovery.

On the basis of this study the following conclusions are offered:

1. Hyperthermic body temperatures decrease the magnitude of the cochlear response. The more severe the temperature deviation, the greater is the loss.

2. Hyperthermia reduces sensitivity, but the form of the intensity function over the greater part of its course is essentially unaltered.

3. No significant recovery was observed following hyperthermia (41° C).

4. The range of body temperature within which the response seems to remain relatively constant is rather extensive (32 to 40° C).

UNIVERSITY OF DELAWARE

REFERENCES

1. Brobeck, J. R.: Regulation of Energy Exchange. In J. F. Fulton (Ed.), A Textbook of Physiology. Philadelphia: W. B. Saunders Co., 1955, (17th ed.), p. 1111.

2. Gulick, W. L., and Cutt, R. A.: The Effects of Abnormal Body Temperature upon the Ear: Cooling. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 69:1:35 (Mar.) 1960.

3. Gosselin, R. E.: Acute Hypothermia in Guinea Pigs. Amer. J. Physiol. 157: 103-115, 1949.

4. Wever, E. G., Bray, C. W., and Lawrence, M.: The Nature of Cochlear Activity After Death. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:317-329, 1941.

LXXIV

THE LOSS OF COUNTER-ROLLING OF THE EYES IN THREE PERSONS PRESUMABLY WITHOUT FUNCTIONAL OTOLITH ORGANS

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The relation between counter-rolling of the eyes and the otolith organs has occupied the attention of many investigators. In man, the results of these investigations have not been uniform and some of the reasons at least are apparent, namely, 1) the impossibility of selectively stimulating the otolith apparatus, 2) the lack of specific indicators of stimulation, 3) inaccuracies in measuring ocular counter-rolling, and 4) the possibility that counter-rolling has multiple causes.

It is generally agreed that the otolith organs respond to linear accelerations but so do many other sensory receptors. Our search for specific indicators of stimulation of the otoliths, other than counter-rolling, has revealed only one test, the oculogravic illusion, which may fulfill this requirement under carefully specified conditions.[1,2] The absence of this illusion in some deaf persons has been known since the classical experiments of Kreidl.[3] However, few investigators have had a human centrifuge at their disposal and, in lieu of this test, have assumed that if there was a loss of function of the semicircular canals, the otolith apparatus also was non-functioning. This raises the question of criteria for loss of function of the canals and the further question of whether loss of function of the canals is a valid test for loss of function of the otoliths.

The difficulties of measuring, accurately, counter-rolling of the eyes are well known to those who have attempted these measurements and are declared by the great variations in their findings.[4-6] In a

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previous report, we have summarized some of this experience and described a new modification utilizing sutures in the conjunctiva as markers.[7] The problem is further complicated, however, by the possibility that there are multiple causes of counter-rolling. Fischer[5] has stated that it may have its origination in reflexes from the cervical region. Finally, the lack of a regular dependence between the angle of tilt and the amount of counter-rolling in healthy persons has discouraged investigators from exploiting the method. However, this point has been resolved by showing that the dependence is regular if the magnitude of lateral force is used as the independent variable.[8]

Despite these limitations and difficulties, it is important to establish the validity of counter-rolling as a specific indicator of otolith stimulation not because the method is good but simply because it is the best practical procedure in sight. What follows is a description of experiments in which counter-rolling was measured in persons who did not perceive the oculogravic illusion and thus presumably had lost the function of the otolith organs.

PROCEDURE

Subjects. Three totally deaf subjects were chosen from a large group. All were highly intelligent, co-operative, and experienced observers.

CASE 1. P.H., a 37 year old woman, became totally deaf at the age of 9 years as the result of a head injury. She had participated in previous experiments and repeated examinations disclosed that she was totally deaf to noise levels up to 155 db; that she did not exhibit nystagmus when the ears were irrigated with ice water for three minutes; and that she never perceived the oculogravic illusion.

CASE 2. D.G., a 27 year old woman, had severe mastoiditis at the age of 4 years requiring bilateral mastoidectomy. Two years later she had a left radical mastoidectomy, followed in another two years by a right radical mastoidectomy. In the course of our examination, she was not exposed to the high intensity noise levels as in the case of the other two subjects but did not perceive loud (uncalibrated) sounds and noise. Nystagmus was not observed following irrigation of the ears with ice water for three minutes, and she did not perceive the oculogravic illusion.

CASE 3. R.G., a 44 year old male teacher, had had spinal meningitis at the age of 3 years after which he was partially deaf. At the age of 12 years, he had a bilateral mastoidectomy after which a hearing

TABLE I

NUMBER OF EXPERIMENTAL TRIALS FOR EACH SUBJECT AT DIFFERENT ANGLES OF TILT AND CORRESPONDING ANGLES ϕ ON THE CENTRIFUGE

	NO. TRIALS, TILT CHAIR							NO. TRIALS, CENTRIFUGE						
	LAT.	PH		DG		RG			LAT.	PH*		DG		RG	
DEGREES	FORCE	R	L	R	L	R	L	DEGREES	FORCE	R	L	R	L	R	L
15	0.26	3	3	3	2	6	6								
30	0.50	3	3	3	2	6	6	30	0.53	0	0	3	2	3	2
43	0.68	3	3	3	0	6	6	43	0.93	0	0	3	0	3	2
66	0.91	3	3	3	2	6	6	66	2.25	0	0	3	2	3	2

* The centrifuge was not in operational condition, hence PH was not tested.

TABLE II

MEAN OCULAR TORSION AND STANDARD DEVIATION FOR ALL EXPERIMENTAL TRIALS FOR ALL SUBJECTS

ANGLE OR SPEED	AVERAGE FOR 5 NORMAL SUBJECTS	D. G.	P. H.	R. G.
10 rpm R (30°)	2.9 ± 1.2	+ 0.6 ± 0.5		+ 0.1 ± 0.2
13 rpm R (43°)	4.8 ± 1.2	+ 1.0 ± 0.9		+ 0.3 ± 0.3
20 rpm R (66°)	8.5 ± 2.8	+ 2.3 ± 1.8		— 0.6 ± 0.8
10 rpm L (30°)	— 3.3 ± 0.8	— 2.8 ± 0.9		+ 0.1 ± 0.5
20 rpm L (66°)	— 8.4 ± 2.9	— 3.3 ± 1.1		+ 2.0 ± 1.0
15° R	1.6 ± 0.7	+ 0.6 ± 0.4	— 0.2 ± 1.6	+ 0.8 ± 0.9
30° R	2.8 ± 1.4	+ 1.2 ± 0.6	— 0.1 ± 1.4	+ 0.6 ± 0.7
43° R	3.2 ± 1.4	+ 1.2 ± 0.5	+ 0.4 ± 0.7	+ 0.6 ± 1.1
66° R	4.2 ± 2.2	+ 1.3 ± 0.6	+ 1.2 ± 0.7	+ 0.3 ± 1.2
15° L	— 2.2 ± 1.4	— 0.4 ± 0.1	— 0.4 ± 0.8	— 0.6 ± 0.3
30° L	— 3.4 ± 1.6	— 1.8 ± 0.4	— 0.6 ± 0.2	— 0.2 ± 0.8
43° L			+ 0.2 ± 0.2	— 0.7 ± 0.3
66° L	— 3.8 ± 1.8	— 1.5 ± 0.2	— 1.1 ± 0.8	— 1.5 ± 1.2

aid was ineffectual. At the age of 21 years, he had a second bilateral mastoidectomy and after that was regarded as totally deaf. He had served as a subject in previous experiments which disclosed that 1) he perceived the oculogravic illusion (July, 1955), 2) he perceived a faint whistle in the left ear at 130 db, and 3) a slight fine nystagmus was observed when his left ear was irrigated with ice water for three minutes. Consequently, at the time of the counter-rolling experiment (June, 1958), he was considered to have a functional otolith apparatus on the left side. Subsequently (July, 1959), he was brought back for another series of experiments, and at that time his threshold for hearing had gone up, nystagmus was no longer perceived on irrigation of the left ear with ice water for five minutes, and he did not perceive the oculogravic illusion under conditions in which healthy subjects perceived the illusion.[9] Whether or not at the time of the counter-rolling experiments he had a residuum sufficient to perceive the oculogravic illusion is not known. Both possibilities will receive consideration.

METHOD

The procedure described in an earlier report was followed.[7] Counter-rolling was determined by comparative measurements of the position of the eye made from photographs of the face before, during, and after each test procedure. Landmarks on the eye were the insertion points of fine silk sutures in the conjunctiva on either side of the limbus. Landmarks on the head were the corners of small patches of black adhesive tape placed near the outer canthus or the natural markings(inner canthus), or both. Lines were drawn on the serial photographs, and the changes in angle corresponding to a rotation of the eye with reference to the head were measured.

Previous observations on healthy persons[8] disclosed that the amount of counter-rolling obtainable was greater on the human centrifuge than on the tilt chair. This same procedure was followed which allowed direct comparison between the two groups of subjects. The experimental trials were set up in such a manner that, although the magnitude of the forces were greater on the centrifuge than on the tilt chair, the incident angles or direction of the forces were kept the same. Table I shows the comparative incident angles and the magnitudes of the lateral force component (which were considered to be the effective stimulus to the otolith organ), the number of experimental trials, and the direction of incident force (right or left). Since Fischer[5] reported that cervical reflexes might stimulate counter-rolling, head braces and supports were used here to maintain head position and to prevent insofar as possible tension on neck muscles.

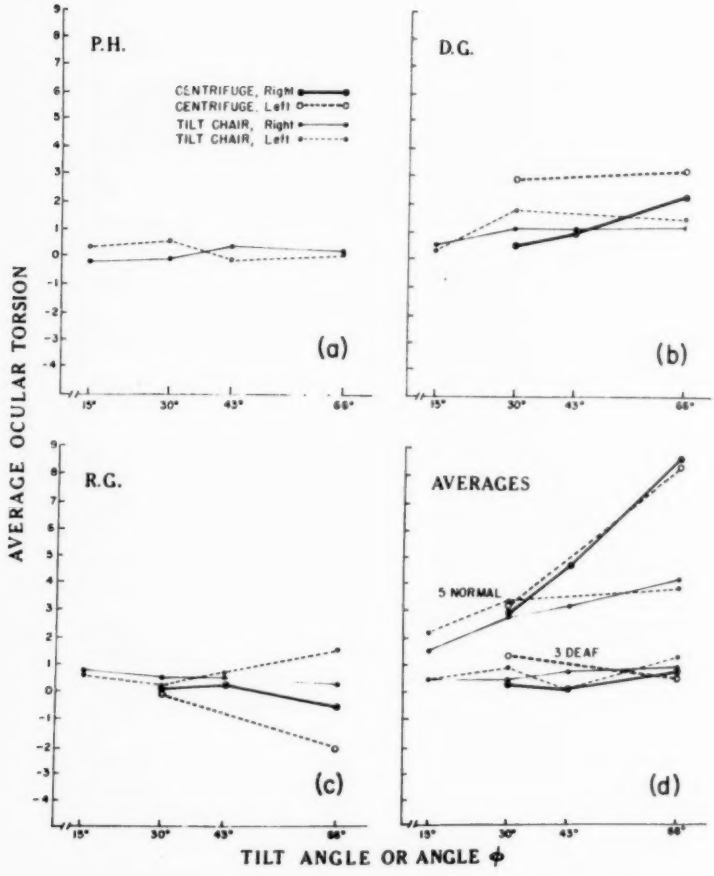

Fig. 1.—Mean ocular torsion during tilt and exposure to change in direction of force on a human centrifuge in the case of 3 subjects (a, b, c) with bilateral labyrinthine defects and (d) a comparison with findings in healthy persons.

RESULTS

The mean and standard deviations of the counter-rolling measurements for the subjects in all conditions are shown in Table II. In Figure 1 the mean torsions for each angle are plotted for each subject and the mean combined values for the three subjects and for the five healthy subjects previously reported.[8] The signs are reversed on the values for tilt to the left (dotted lines) so that ocular torsion in opposite directions resulting from right and left stimuli lies on the same side of the baseline for comparison purposes.

COMMENT

The greater variation in values obtained in the experiments carried out on the human centrifuge compared with the tilt chair may represent the somewhat greater technical difficulty or possibly the influence of some other factor such as tension on the neck muscles. It is worth noting that the error on the centrifuge is consistent, i.e., plus when direction is right and minus when direction is left.

The great difference between the values obtained for the healthy and deaf subjects is striking. To determine the statistical validity of this difference, the counter-rolling at the various values of right tilt for each subject was compared with the normal group by the use of the F test of variance. In each case, the difference was significant at the .05 level. The difference between persons with and without labyrinths is even more striking when the values for torsion under increased lateral force on the centrifuge are compared. Since the ocular torsion in normals increases with increasing lateral force, any counter-rolling at all in the deaf would be expected to appear at the highest forces on the centrifuge. It is seen, however, that there is no consistent increase in torsion with the larger forces, and that even the maximum stimulus produces no significant counter-rolling reflex in the deaf subjects.

The absence of counter-rolling in R.G. deserves particular mention because there was a possibility he could perceive the oculogravic illusion. In this event, it must be concluded that a residuum of function sufficient for perception of the oculogravic illusion was insufficient for initiating the counter-rolling reflex. That this is a reasonable explanation is suggested by research on the fine structure of the macula disclosing important regional differences.[10]

These findings strongly suggest, if they do not prove, that counter-rolling is a response to stimulation of the otolith organs. They

do not eliminate the possibility that counter-rolling might be due to other causes, viz., visual factors, neck reflexes, and possibly other factors. Inasmuch as there is a very great need for specific indicators of otolith function, this line of investigation should be pursued.

U. S. Naval School of Aviation Medicine

We are sincerely grateful to Doctor Charles D. Sneller for making Case 2 available to us together with his findings.

REFERENCES

1. Graybiel, A.: Oculogravic Illusion. Arch. Ophthal. 48:605-615, 1952.

2. Graybiel, A.: The Importance of the Otolithic Organs in Man Based Upon a Specific Test for Utricular Function. Annals of Otology, Rhinology and Laryngology, St. Louis, 65:470-488, 1956.

3. Kreidl, A.: Beiträge zur Physiologie des Ohrlabyrinths auf Grund von Versuchen an Taubstummen (Contributions to the Physiology of the Labyrinth of the Ear on the Strength of Experiments on Deaf-mutes). Pflüg. Arch. ges. Physiol. 51: 119-150, 1892.

4. Barany, R.: Über die vom Ohrlabyrinth ausgelöste Gegenrollung der Augen bei Normalhorenden, Ohrenkranken und Taubstummen (Concerning the Counter-rolling of the Eyes Released from the Labyrinth of the Ear in Subjects with Normal Hearing, in Patients with Diseases of the Ear and Deaf-mutes). Arch. Ohr.-Nas.-u. Kehlkheilk 68:1-30, 1906.

5. Fischer, M. H.: Messende Untersuchungen Ueber die Gegenrollung der Augen und die Lokalisation der scheinbaren Vertikalen bei seitlicher Neigung des Gesamtkörpers bis zu 360°. II. Mitteilung (Comparative Investigations on the Counter-rolling of the Eyes and the Localization of the Apparent Vertical in the Lateral Inclinations of the Whole Body up to 360°. II. Communcation). v. Graefes Arch. Ophthal. 123:476-508, 1930.

6. Gollas, E.: Untersuchung der Raddrehung der Augen bei einseitigem Labyrinthausfall (Investigation of the Torsion of the Eyes in a Unilateral Absence of the Labyrinth). Arch. Ohr.-, Nas.-, u. KehlkHeilk 140:340-354, 1936.

7. Graybiel, A., and Woellner, R. C.: A New and Objective Method for Measuring Ocular Torsion. Am. J. Ophthal. 47:349-352, 1959.

8. Woellner, R. C., and Graybiel, A.: Counter-rolling of the Eyes and Its Dependence on the Magnitude of Gravitational or Inertial Force Acting Laterally on the Body. J. Appl. Physiol. 14:632-634, 1959.

9. Graybiel, A., Clark, B., and Zarriello, J. J.: Observations on Human Subjects Living in a "Slow Rotation Room" for Periods of Two Days. Archives of Neurology (to be published).

10. Smith, C. A.: Microscopic Structure of the Utricle. Annals of Otology, Rhinology and Laryngology, St. Louis, 65:540-469, 1956.

LXXV

CARCINOMA OF THE LARYNX: COMPARATIVE EXPERIENCE IN THE MANAGEMENT OF 951 LIMITED LESIONS

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A small, localized squamous cell carcinoma on the edge of a vocal cord is more amenable to cure than any other cancer except a basal cell carcinoma of the skin. This is of course largely by virtue of early symptomatology, ease of visualization, and the paucity of lymphatics in the vicinity of the true cords. The high salvage rate of carcinoma of the larynx in general is due in great measure to the sustained high five year cures rates (70 per cent to 95 per cent) in cases of small cancers of the true cords. These figures are fantastically high for any kind of cancer.

The chief problem with the large lesion is not how to refine an operation to encompass it, but in the education of practitioners thus permitting diagnosis of the lesion while it is still small enough to fall into the high cure rate bracket. The number of total laryngectomy failures will decrease only with proportionate increase in patient and physician education. Five-year cancer cures by total laryngectomy at the University Medical Center have remained almost static over the past 20 years, regardless of innovations such as wide-field over narrow-field laryngectomy, which makes for a technically nicer operation. Narrow-field laryngectomies performed from 1936 to 1945 produced a 50.0 per cent five-year cure rate. During that time 268 patients were treated for cancer of the larynx. During the next seven years, 1946 through 1952, 265 patients were treated. Wide-field laryngectomy was then done in almost all instances where laryngectomy was indicated, and a 48.5 per cent five-year cure rate resulted. This experience is similar to that at other institutions.

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The kind of laryngeal cancer that is most likely to be cured by today's methods is the small glottic carcinoma—i.e., a lesion on the true cord reaching neither the anterior commissure nor the vocal process, and not impairing cord mobility. There is common agreement at this time that irradiation can produce results equal to surgical treatment of this lesion. Our experience has generally borne this out. Since 1946, the Department of Radiation Therapy has treated an increasing number of limited (sometimes called Stage 1, beyond carcinoma-in situ) lesions, so that today results of irradiation and surgical removal are comparable. From 1946 to 1952, 37 of such limited lesions were seen, 14 of which were treated by irradiation and 23 by operation. Five-year cures resulted in 83% (maximum or indeterminate,* 91%) of surgical cases, and in 78% (maximum or indeterminate, 86%) of irradiation cases. The experiences of other laryngologists and radiotherapists in dealing with the same lesion were determined from published reports (Table I).

TABLE I

LIMITED LARYNGEAL LESION: COMPARATIVE RESULTS

SURGERY	*Period in Yrs.*	*Cases*	*Result*†	X-RAY	*Period in Yrs.*	*Cases*	*Result**
Martin (1947)	10	21	95%	Baclesse (1951)	6	11	91%
LeJeune (1958)	..	67	95%	Neilsen (1951)	15	18	89%
New (1946)	10	184	84%	Harris (1951)	16	20	65%
Tucker (1952)	13	152	86%	Lenz (1947)	..	10	80%
Jackson (1948)	17	102	81%	Lederman (1952)	..	12	83%
Colledge (1945)	..	23	83%	Schall (1948)	15	11	46%
Schall (1948)	10	31	58%	Simson Hall (1953)	10	12	66%
Simson Hall (1953)	10	10	90%	Jacobson (1952)	10	15	80%
Hoover (1953)	..	23	80%	Binkley (1953)	4	17	60%
Lynch (1955)	..	..	86%	Harmer (1953)	28	31	87%
Mattick (1954)	9	17	83%	Wang (1955)	20	35	80%
Wang (1955)	20	15	87%	Low Beer (1950)	5	19	79%
U of M (1959)	7	23	83%	Cutler (1950)	7	58	71%
				U of M (1959)	7	14	78%
Total cases:		668		Total cases:		283	
Average 5 year cure rate: 83%				Average 5 year cure rate: 75%			

† Results are in each case determinate 5 year cures.

* Counting as cured, patients dying without evidence of cancer in less than five years.

The above table surveys over 900 cases of the limited lesion and probably reflects significantly world-wide experience with this particular lesion. Irradiation produced five-year cures in three out of four patients, while surgery cured better than four out of five. The best x-ray results are however comparable to the best surgical results. This suggests that where the services of a highly competent radiotherapist are available, he may offer the patient with a limited lesion a chance equal to the surgeon. Whether the irradiation is delivered by high voltage machines or radium applicators seems to make little difference, if the author is skilled in its delivery. The Finzi-Harmer fenestration method of radium needling, for example, produces excellent results in the hands of Finzi and Harmer.

Whatever method of irradiation is selected, it must further be borne in mind that it is not a conservative form of treatment. It is fraught with risks. Results are bought not without considerable discomfort from the usual epidermitis and mucositis which is often prolonged. It must be administered with daily care, which must include daily indirect laryngoscopy. One point which is often forgotten is that a good serviceable voice, even upon control of the tumor, cannot be guaranteed. One patient might be more hoarse following irradiation than might another patient following hemilaryngectomy. It is equally true, however, that some patients tolerate cancericidal doses of x-ray with few after-effects. This is especially true of the patient with the limited lesion. We have seen no cases of radiation chondritis in this classs, and an occasional patient is seen whose larynx three months after x-ray therapy defies detection of radiation effects.

If the services of an expert radiotherapist are not available, suitable operation provides a high likelihood of survival. If during radiotherapy it becomes evident the tumor is not being controlled, there exists a demand indication for operation.

There is an occasional patient with a limited lesion for whom either hemilaryngectomy or irradiation may constitute over-treatment. Furstenberg has remarked on the importance of evaluating the clinical behavior of a cancer. He has long felt nothing is lost taking a little time to ascertain the clinical course of a doubtful lesion. This principle has been instrumental in producing a 100 per cent cure rate in a selected group of patients. The treatment carried out (save in one case) has been solely direct laryngoscopy and cord scalping, or repeated cord scalping, and close observation. The superficial substance of the cord was in each case widely removed by biopsy forceps down

TABLE II

LIMITED LARYNGEAL LESION: TREATMENT BY LARYNGOSCOPIC TECHNIQUES

PATIENT	APPEARANCE OF LESION	TREATMENT	PATHOLOGY REPORT	RESULT
1.	Fibrous mass junction ant. and mid ⅓ left true cord. Cord mobile	Removal with forceps via laryngoscope	Squamous cell carcinoma with evidence of infiltrative growth	Alive without disease 11 9/12 yr.
2.	Papillomatous growth ant. commissure and ant. extremity rt. true cord	Removal with forceps via laryngoscope	Squamous cell carcinoma in a papilloma, which may have developed in the papilloma	Dead without carcinoma 3 5/12 yr.
3.	Nodule in mid ⅓ left true cord. Cord mobile	Removal with forceps via laryngoscope	Slightly cornifying squamous cell carcinoma, grade II	Recurrence, Reoperate, X-ray. Alive 5 yr.
4.	Constructive lesion left true cord. Cord mobile	Removal with forceps via laryngoscope	Cornifying squamous cell carcinoma	Alive without disease 8 9/12 yr.
5.	Polypoid mass ant. ½ both true cords, more on right. Cords mobile	Direct laryng. and cord scalping	Papillomatous hyperplasia of squamous epithelium with early invasive growth (sq. cell ca, II)	Alive without disease 8 9/12 yr.
6.	Polypoid mass ant. commissure and immediate subglottic region. Mob.	Removal with forceps via laryngoscope	Cornifying squamous cell carcinoma with shallow infiltration	Alive without disease 7 2/12 yr.
7.	Sessile papillomatous nodule right true cord. Cord mobile	Direct laryng. and cord scalping	Squamous cell carcinoma	Alive without disease 7 2/12 yr.
8.	Constructive pink mass anterior ⅓ left true cord. Cord mobile	Removal with forceps via laryngoscope	Cornifying squamous cell carcinoma with infiltration	Alive without disease 7 years
9.	Multiple vegetative papillomata both true cords. Cords mobile	Direct laryng. and cord scalping	Two fragments show cornifying squamous cell carcinoma with shallow infiltration	Alive without disease 6 years
10.	Constructive lesion mid ⅓ right true cord. Cord mobile	Two laryngoscopies with cord scalping	Widespread infiltrating cornifying squamous cell carcinoma. (2nd: presumptive diagnosis of carcinoma)	Alive without disease 5 years

to the substantia propria of the true cord, without disturbing the fibrous substance of the cord. Since 1936, 513 patients with laryngeal cancer received definitive treatment. From 1946 through 1952, 265 patients were treated, of which 37 possessed a limited lesion. Of these 37, 4 were managed by local excision. Of the limited lesions seen in the previous ten years, 6 were so treated. Since 1952, 2 additional patients are alive without disease, but treated recently enough not to be accountable five-year cures. The type of lesion amenable to this form of treatment has constituted roughly 10 per cent of all limited lesions. These cases are of particular interest because they demonstrate that conservatism may still play a distinct role in the successful management of cancer.

A factor which may be of importance in the high cure rate in this selected series is the evident high degree of host resistance to the tumor. Each remained localized and superficial through the periods of growth, diagnosis and treatment; each was one of those carcinomas by its very behavior highly amenable to cure. We have all seen carcinomas which proliferate so wildly that radical treatment instituted even a few weeks after onset of symptoms contains it but momentarily and the patient rapidly succumbs. There must exist a biochemical and biophysical difference between tumors which is not reflected in morphological changes, e.g., Broder classification. This difference may even exist between parent and daughter cells of the same tumor: the massive cervical metastases from a scarcely detectable nasopharyngeal primary serves as an excellent example. We know but little about the nature of this phenomenon, but clinical use can be made of this behavioral dependence of carcinoma on host resistance.

The third patient in the tabulated series is the only patient in the series to receive more than treatment by cord scalping. Upon routine close observation, he was found two months later to have a suspicious area developing at the site of his previous operation. This proved to be a recurrence. He was subjected to irradiation and is alive without disease almost ten years later. Although this is a single case, it raises the interesting possibility that strict true cordal carcinoma, while still in its early stages, may act in a biologically different manner than carcinomas of other areas of the endolarynx poor in lymphatics: the accepted dictum that "the first person to treat the lesion has the best chance to cure" did not hold here. After two surgical attempts, irradiation effected a cure.

Critical interpretation of a doubtful pathology report may yield dividends. The tenth patient was observed meticulously after his

second cord scalping specimen was read as showing epithelial changes "requiring the presumptive diagnosis of carcinoma." The temptation to advise x-ray at this point is strong. His larynx had not the clinical appearance of carcinoma, however, and observation alone was carried out over the next six months. Persistent roughening of the surface of the cord dictated repeat cord scalping, and this proved to be atypical epithelial hyperplasia. Upon resurfacing, the cord maintained a normal appearance.

The above patients as a whole, of course, tended to have better voices and more normal appearing larynges than patients following either irradiation or hemilaryngectomy. Finally, it must be reiterated that the above form of treatment is an election possible in only a small number of limited lesions, for patients in whom adequate follow-up is virtually guaranteed.

SUMMARY

The highest cure rates obtainable by today's methods for the management of laryngeal cancer are in the treatment of the small glottic carcinoma. Both surgery and x-ray offer excellent results, and today the latter is the preferred form of therapy if this is administered by a competent radiotherapist who observes the larynx daily. This of course does not apply in those communities where the general radiologist in a small hospital with limited radiation facilities manages the occasional therapy case. The number of such radiation specialists is small country-wide.

It is possible to effect, in a small but significant number of patients with a limited lesion, a cure purely by laryngoscopic techniques.

While surgery and radiation therapy remain our only effective tools for the extirpation of cancer of the larynx, our cure rates will rise only by virtue of use of the laryngeal mirror by every practitioner of medicine, particularly the general practitioner.

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REFERENCES

1. Martin, Hayes: Cancer of the Larynx. Surgery of the Nose and Throat. Edit. by J. D. Kernan, 431-504, Nelson and Sons, N. Y., 1947.

2. LeJeune, F. E.: Personal Communication.

3. New, G.: Carcinoma of the Larynx: Methods of Treatment and Results. President's Address, Trans. Amer. Acad. Ophth. and Otolaryngol. 51:7-10 (Sept.-Oct.) 1946.

4. Tucker, G.: Diagnosis and Treatment of Cancer of the Larynx. J.A.M.A. 149:119-121 (May 10) 1952.

5. Jackson, C. L., Blady, J. V., Norris, C. M., and Maloney, W. H.: Cancer of the Larynx. J.A.M.A. 138:1080-1083 (Dec. 11) 1948.

6. Colledge, L.: Discussion on Treatment of Carcinoma of the Larynx. Proc. Roy. Soc. Med. 38:354-357 (May) 1945.

7. Schall, L. A., and Ayash, J. J.: Cancer of the Larynx: Statistical Study. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:377-386 (June) 1948.

8. Simson, Hall, I.: Conservative Treatment of Cancer of the Larynx. J. Laryngol. and Otol. 67:203-214 (Apr.) 1953.

9. Hoover, W. B.: Carcinoma of the Larynx: Overall Results and Trends in Treatment. Surg. Clinics of North America (Lahey Clinic No.) 805-812 (June) 1953.

10. Lynch, M. G.: Prognosis in Laryngofissure. Trans. Amer. Laryngol. Assn. 76:107-113, 1955.

11. Mattick, W. L., Mancini, A. J.: Intrinsic Carcinoma of the Larynx. Arch. Otolaryngology 59:71-75 (Jan.) 1954.

12. Wang, C. C., and O'Donnell, A. R.: Cancer of the Larynx: Five Year Results, With Emphasis on Radiotherapy. New England J. Med. 252:743-747 (May 5) 1955.

13. BaClesse, F.: Roentgentherapy of Carcinoma of the Larynx. J. Fac. Radiol. 3:3-12 (July) 1951.

14. Nielsen, J.: Functional Results and Permanence of Cure Following Roentgentherapy of Intralaryngeal Carcinomas. J. Fac. Radiol. 3:29-34 (July) 1951.

15. Harris, W.: Roentgentherapy for Cancer of the Larynx. J. Fac. Radiol. 3:35-39 (July) 1951.

16. Lenz, M.: Roentgen Therapy in Cancer of the Larynx. J.A.M.A. 134:117-121 (May 10) 1949.

17. Lederman, M.: Some Problems in Radiation Treatment of Cancer of the Larynx. Annals Roy. Coll. Surg. of Eng. 11:47-61 (July) 1952.

18. Jacobsson, F.: Teleradium Treatment of Laryngeal Carcinoma. Acta Radiologica 38:143-161 (Aug.) 1952.

19. Binkley, J. S.: Carcinoma of the Larynx Treatment by 500 KV Roentgen Rays. Amer. J. Roentgenology 70:591-598 (Oct.) 1953.

20. Harmer, W. D.: Treatment of Cancer of the Larynx by Interstitial Radium Needles. Brit. Med. J. 2:735-741 (Oct. 3) 1953.

21. Low Beer, B. V. A.: Radiation Therapy of Cancer of the Larynx. Laryngoscope 60:696-717 (July) 1950.

22. Cutler, M.: Radio Therapy of Early Cancer of the Larynx: Five Year Results. J.A.M.A. 142:957-963 (Apr. 1) 1950.

LXXVI

DIAGNOSTIC APPLICATIONS OF RADIOPHOSPHORUS FOR MALIGNANT NEOPLASMS IN OTORHINOLARYNGOLOGY

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It has been widely known for many years that the radiation emitted from radioisotopes selectively deposited in tissue may be used to record the geometric disposition of these elements. Radioactive phosphorus administered intravenously in a living body is assimilated by different tissues in varying degrees. The concentration of P^{32} in tissue at any given time after administration depends on the metabolic activity of the constituent cells. Since the earlier investigation of Hevesy,[1] Low-Beer[3] et al, it is undoubtedly recognized that malignant neoplasms have a higher metabolic rate of P^{32} than the normal tissue from which they originate. Therefore, diagnostic applications of radiophosphorus for malignant neoplasms have been rapidly popularized in clinical medicine, as earlier reported.[3,5,6] Nevertheless, in the otorhinolaryngological field, there is no such systematic investigation which has incorporated radiological foundation with the clinical applicability of the radiophosphorus. With this in mind, the author has undertaken the following investigation.

PHOSPHORUS DISTRIBUTION IN NORMAL AND CANCEROUS HUMAN TISSUE

In order to confirm the theory that growing human neoplasm might show a higher and more rapid uptake of P^{32} than other normal tissue, the author investigated phosphorus distribution in various human tissues and compared the radio-activity of normal tissue with that of malignant neoplasms in otorhinolaryngology.

Material and Method. The author used disinfected physiological saline solution of orthophosphate $P^{32}O_4$ in dilute hydrochloric acid imported for clinical applications from the Radiochemical Centre,

From the Department of Otorhinolaryngology, School of Medicine, Kyoto University.

Amersham, England, and Oak Ridge National Laboratory, U.S.A. Subjects were 78 in-patients of the Department of Otorhinolaryngology of Kyoto University Hospital. Intravenous injections of P^{32} solution (on the average of 300 microcuries) were applied to them pre-operatively. The details are shown in Table I.

Scaling samples taken from the various parts of the human body by means of operation, exploratory excision, or autopsy were weighed and dissolved by wet ashing. Inactive potassium phosphate was added to them and the phosphorus was precipitated as magnesium ammonium phosphate by using Hevesy's method.[4] Fresh samples of about 1 to 0.5 gm were found to be convenient. P^{32} labeled magnesium salt was precipitated in numerous punched tin dishes. Activity of radiophosphorus was calculated by the Shimadzu "1000" Scaler D-55A connected with the Philips No. 18506 Geiger-Müller Tube.

Result. Quantitative measurement of radioactivity of each sample allowed calculation of the "differential absorption ratio" of various tissues as defined by Marinelli et al[1] (Table II).

Also, the phosphorus distribution in cancer-bearing human bodies was assayed. In the case of lingual squamous cell cancer, when P^{32} (12.1 microcuries per Kg of body weight) was administered intravenously, the pieces of tissue assayed were obtained within 208 hours after the administration of the P^{32}. The radioactivities of autopsied tissues, expressed in micromicrocuries per gram wet weight, were as follows: liver 3080, spleen 2760, bone 2550, lung 1970, kidney 1690, pancreas 1670, primary tumor 1070, stomach 972, aorta 898, heart muscle 829, uterus 699, thyroid gland 674, small intestine 533, etc. In a patient with maxillar squamous cell cancer from which she died shortly after the operation, the pieces of neoplastic tissue retained more radiophosphorus than did those more rapidly metabolizing organs such as the liver and spleen. In this case intravenous administration of P^{32} was 7.68 microcuries per Kg of body weight and the time between the P^{32} injection and autopsy was 26 hours. The radioactivities of various tissues were as follows: marginal area of neoplasm 3530, central area of neoplasm 3210, liver 2990, spleen 2600, pancreas 2060, suprarenal gland 1920, thyroid gland 1690, heart muscle 1380, submandibular gland 947, ovary 637, etc.

The geographic distribution of various areas of neoplastic tissue was demonstrated by macro- and micro-autoradiography employing P^{32}. A block specimen of the maxillar region containing cancerous tissue was sectioned in five slices of about 1 cm thick, photographed,

TABLE I

Malignant neoplasms	
Maxillar cancer	27
Laryngeal cancer	8
Pharyngeal cancer	7
Cancer of the oral cavity	6
Pharyngeal sarcoma	5
Cervical esophageal cancer	4
Nasal and paranasal sarcoma	2
Sarcoma of the lower jaw	1
Benign neoplasms	
Adenoma of the neck and its vicinity	3
Angioma of the maxilla and tongue	2
Cyst of the upper and lower jaw	2
One-sided chronic inflammations	
Paranasal sinusitis	7
Other chronic E.N.T. inflammations	4
Total	78

after coming into contact with the Fuji medical x-ray film was exposed in a cold dark place for about 90 hours.

Note that the neoplastic tissue is much denser than the other surrounding normal tissue, also that the necrotizing area near the center of the tumor is much less dense than the marginal area of neoplastic tissue.

In conclusion, the majority of the tissues of malignant neoplasm or those infiltrated with malignant tumor cells retained as much or more radiophosphorus than other rapidly metabolizing tissues of the same patient.

CLINICAL STUDY

There are some difficulties in utilizing radiophosphorus as a diagnostic indicator for malignant neoplasms by superficial reading of the radioactivity. That is to say, the most penetrating beta particles emitted from P^{32} have an energy of 1.7 MeV, namely, a range of 8 mm of soft tissue. The superficial reading of the radioactivity of a tumor located in subcutaneous tissue was influenced chiefly by the

TABLE II

DIFFERENTIAL ABSORPTION RATIO OF RADIOACTIVE PHOSPHORUS FOR PATIENTS WITH MALIGNANT NEOPLASMS IN OTORHINOLARYNGOLOGY AND CONTRAST CASES

CASE NO.	P^{32} (μc) ADMINISTERED PER KG OF BODY WEIGHT	HOURS BETWEEN P^{32} ADMINISTRATION AND OPERATION	D.A.R. IN VARIOUS TISSUES									
			PRIMARY TUMOR	SKIN	MUCOSA	FAT	MUSCLE	BONE	TEETH	CARTILAGE	GLAND	LYMPHNODE
2	6.74	21.5	1.25		0.61		0.58	0.44				
22	7.34	4	2.60			0.79	0.75	1.20				2.47
27	7.69	8	2.53		1.83	0.31	0.10	3.62				
43	9.09	4	3.23		1.28	0.34	0.82	1.74	0.62			2.36
41	26.3	7	6.01					3.21		1.96		
44	20.6	3.5	3.80		1.85		0.90	2.04		1.69	1.35	
47	27.4	4	4.14		2.99		0.81	1.72		0.93		2.98
4	5.45	22	2.10	1.05		0.19	0.68	0.57				0.99
26	6.00	4.5	4.18				0.71	1.24		2.28	1.33	
29	7.69	4	3.69		2.39		1.46					3.77 (metastatic)
37	21.5	5.5	1.23	0.48			0.41		1.74			
42	24.0	4	3.75				3.08	6.44				6.01 (metastatic)
50	22.4	4	6.79				1.16	5.75	5.20		4.58	4.40
51	24.1	5	9.49	1.46			4.34	5.72				4.46
28	6.74	5	0.69 (cyst)				0.14	0.31	0.18		0.53	
76	8.46	96	0.38 (hemangioma)		2.02	0.27		4.18				
30	6.82	8			2.05)			2.30				
36	7.21	5			3.49)		1.41	4.80				
39	6.32	4			1.29)	0.32	0.70	3.13				
					(chronic inflammation)							

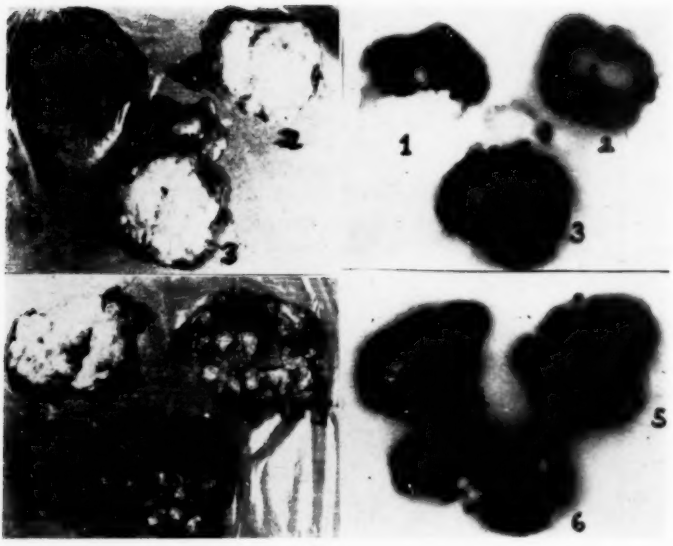


Fig. 1.—Macro-autograph of maxillary squamous cell cancer.

depth from the skin surface. In order to make a differential diagnosis in otorhinolaryngology between normal and cancerous tissue of a living patient, the examiner should make contact between the G.M. tube and the suspected area as close as possible, and then compare it with the radioactivity of the same tissue on the normal side.

Material and Method. The author used three miniature counter tubes of halogen quenched type made by Anton Electronic Laboratory, U.S.A. The type 155, an all stainless steel rugged and needle-shaped thin wall subminiature probe counter tube (diameter 2.5 mm, wall thickness 30 mg/cm^2) was used for the measurement of the nasal and pharyngeal cavity. The type 225 was a highly sensitive subminiature probe counter tube having a pancake designed end mica window, and curved in order to afford greater accessibility to the epi- and hypo-pharynx (mica window diameter 4.8 mm, thickness 1.4-2.0 mg/cm^2). The type 244 was also a highly sensitive miniature counter tube designed with a long axis to permit very accurate examination of the oral cavity and skin surface (mica window diameter 6.4-9.5 mm, thickness 1.4-2.0 mg/cm^2) (Fig. 2).

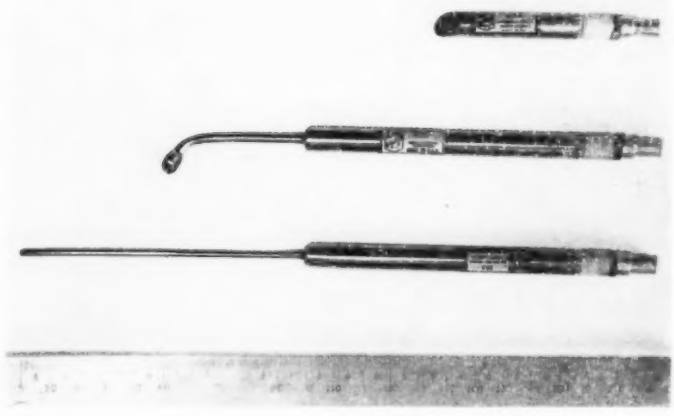


Fig. 2.—Miniature counter tubes for tumors in otorhinolaryngology.

Each counter tube was connected with the Shimadzu A-12 preamplifier and "1000" scaler D-55. Other techniques were all the same as the preliminary study.

Result. P^{32} activity in the skin surface on both the normal and the diseased side was measured as a function of time. Generally speaking, the concentration ratio between counts per minute of tumor infiltrated area and of opposite normal skin amounted to the minimum value in 10 to 30 minues following intravenous administration of P^{32}, and the maximum value appeared about three hours later (Fig. 3).

Black spots indicate the concentration ratio of malignant neoplasm and white spots indicate the same for those which are considered normal.

For tumors of the nasal, oral, epipharyngeal and hypopharyngeal cavity, where the author was unable to determine the grade of malignancy by superficial reading from the skin surface, he measured the radioactivity of the tumor in situ by inserting directly one of the above-mentioned miniature counter tubes into those cavities and then comparing the radioactivity of the tumor with that of the corresponding opposite normal mucous membrane. Eight cases diagnosed early by this counting technique before histological determination are shown in Table III.

TABLE III

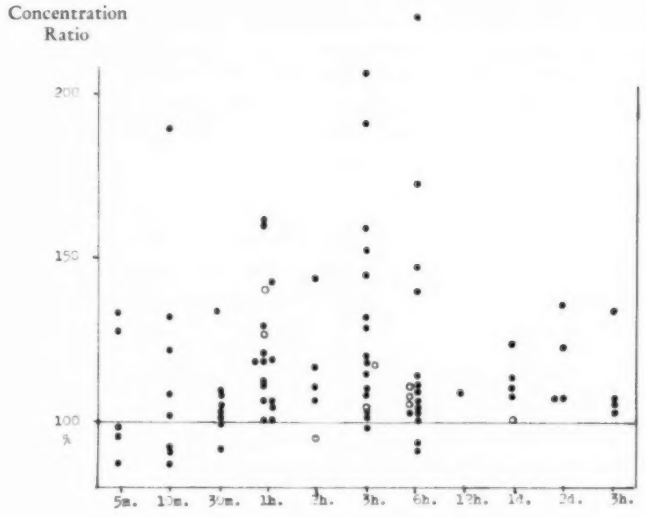
CASE NO.	INVOLVING AREA	HISTOLOGICAL FINDING	CONCENTRATION RATIO 1 h.	3 h.	6 h.	DIAGNOS.
56	Root of tongue	Cystic lymphangioma	80	86	90	Benign
57	Epipharynx	Reticulosarcoma	219	207	163	Malign.
59	Epipharynx	1) Squamous cell carcinoma 2) Chronic inflammation	139	140	110	Malign.
63	Maxillary region	Chronic inflammation	98	93	107	Benign
67	Hard palate	Mucoepitheloid cell carcinoma	192	142	166	Malign.
74	Epipharynx	Carcinoma simplex	144	162	122	Malign.
75	Maxillary region	Squamous cell carcinoma	143	160	148	Malign.
76	Maxillary sinus	Cavernous hemangioma	75	80	63	Benign

Observing the concentration ration in the skin surface, the author divided the subjects into four groups. The first group consisted of patients whose skin surfaces were infiltrated by malignant neoplastic tissue. The second one was composed of patients with malignant neoplasm located closely beneath the nonaffected cutaneous tissue. The third one was also made up of patients with malignant neoplasm located on a layer still deeper from the skin surface and the fourth group was composed of benign neoplasm and one-sided chronic inflammation cases (Fig. 4).

In the first group radioactivity emitted at points of skin surface over the tumor area was 100 to 200 per cent higher than over the corresponding area of the opposite side. In the second group this concentration ratio extended from 200 to 130 per cent. In the third and fourth group each ratio was less than 130 per cent

Accordingly, it is impossible by means of skin surface measurement of P^{32} radioactivity to discriminate clearly between malignant neoplasms located at a deep layer under the skin surface, and benign neoplasms or one-sided chronic inflammations.

The author then concluded that, if the mean concentration ratio between involved area and normal mucous membrane after 1, 3 and 6 hours following P^{32} administration amounted to more than 130 per



Time Elapsed After Administration of P^{32}

Figure 3

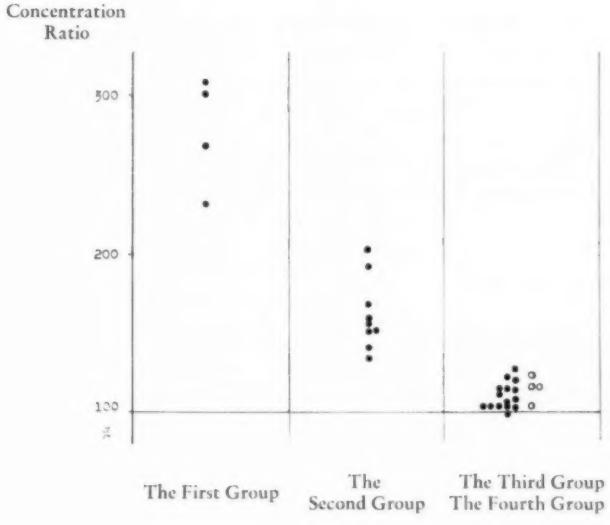


Figure 4

cent, this area could probably be diagnosed as malignant neoplasm. This direct counting technique was similarly adopted in the course of tumor operations to detect malignant tissue in the operation field. To determine recidivation of malignant neoplasm the above-mentioned standard of concentration ratio was also applicable. It seemed that the metastatic lymph node located closely beneath the skin tissue was highly detectable by this technique.

SUMMARY

The author made a fundamental and clinical investigation utilizing radiophosphorus P^{32} as a diagnostic indicator for malignant neoplasms in otorhinolaryngology.

In the preliminary study phosphorus distribution in normal and cancerous tissue was observed by measuring their radioactivity by the calculation of differential absorption ratio and the radioautographic technique. The majority of the tissues bearing malignant neoplasms absorbed radiophosphorus much more abundantly than the other normal tissues of the same patient.

For the purpose of clinical application of P^{32} the author administered intravenously P^{32} saline solution on the average of 300 microcuries to 78 in-patients prepared for radical operation. He then placed Anton's miniature Geiger-Müller counter tubes in contact with the surface and inserted them directly into the body cavity of the subjects. The radioactivity of the involved area and the corresponding normal area was measured and compared at 1, 3 and 6 hours following P^{32} administration. The author concluded that a mean concentration ratio beyond 130 per cent could signify the existence of malignant neoplasm. This superficial counting technique might be also applicable during the course of and after a radical operation for malignant tumor.

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REFERENCES

1. Marinelli, L. D., et al.: Tracer Studies with Radioactive Phosphorus in Malignant Neoplastic Disease. Radiol. 37:683, 1941.

2. Marinelli, L. D., and Goldshmidt, B.: The Concentration of P^{32} in Some Superficial Tissues of Living Patients. Radiol. 39:454, 1942.

3. Low-Beer, B. V. A.: Measurement of Radioactive Phosphorus in Breast Tumors in Situ; a Possible Diagnostic Procedure. Radiol. 47:492, 1946.

4. Hevesy, G.: Radioactive Indicators. Their Application in Biochemistry, Animal Physiology and Pathology. Interscience Pub. Inc., New York, 1948.

5. Selverstone, B., and Steinberg, D.: Radioautography of Cerebral Tumors Employing P^{32}. Proc. Soc. Expl. Biol. and Med. 74:304, 1950.

6. Krohmer, J. S., et al.: Clinical Use of Radioisotopes. A Manual of Technique. Year Book Pub. Inc., Chicago.

7. Nakayama, K.: Diagnostic Significance of Radioactive Isotopes in Early Cancer of the Alimentary Tract, Especially the Esophagus and Cardia. Surgery 39:736, 1956.

8. United Nations: Proceedings of the Second United Nations International Conference on the Peaceful Use of Atomic Energy. Vol. 22-26, Geneva, 1958.

LXXVII

ULTRASOUND IN EAR, NOSE AND THROAT DISEASES

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Pasadena, Calif.

The first paper dealing with the medical application of ultrasound[17] reported experiences in the field of otology. Since that time the use of this modality in otosclerosis, tinnitus, nystagmus, and Ménière's syndrome has been repeatedly discussed in literature. Among the most recent contributions in this use are papers by Altmann and Waltner,[2,3] and Ironside and Lindsay.[11] Most of the papers, as those mentioned above dealing with ultrasonic treatment of disturbances of the middle and inner ear, aim at destruction of the balance center or others tissues or both, and therefore require high dosages of destructive ultrasound.

The present report, in contrast, is concerned with results obtained with low, therapeutic doses of ultrasound applied by means of a multiple crystal sound head which delivers a uniformed distribution of pulsed plane wave energy with freedom from hot or cold spots, in a uniform and easily controlled manner. This improved technique permits the application of ultrasound in a number of conditions commonly encountered in otorhinolaryngology, in which treatment had formerly been at best unreliable, and in some cases of questionable value.

In a strict sense, therefore, many earlier experiences with ultrasound therapy are not applicable, as in some instances a strong central beam when improperly used could have possible dangerous side effects. However, in experienced hands the single crystal sound head with a spot central beam produces excellent therapeutic results when the sound head is properly moved. As a background to the present report, however, the more important contributions to the therapeutic use of ultrasound in otorhinolaryngology (excluding the middle and inner ear) are briefly reviewed.

In 1950 Rosenthal[23] applied ultrasound to various tissues, including the cheek. He also reported dramatic relief of pain from "micromassage" in acute cervical lymphadenitis. Edmundson[8,9] collected a series of some 400 cases in which ultrasonic therapy resulted not only in a clinical improvement of sinus infection, but also in clearing of densities, as demonstrated by roentgenograms. He used a moving sound head at eight watts per square centimeter for one minute (average), and seven watts per square centimeter for three minutes providing localized spot heating. Denier[7] reported excellent results in the treatment of frontal and maxillary sinusitis in cases which had previously failed to respond to antibiotic therapy, or puncture or both. He also recommended ultrasound for spasmodic and allergic rhinitis as well as occipital and facial neuralgia. Krejci[13] had good results with ultrasound treatment of trigeminal neuralgia of peripheral origin. Subacute cases required four to five treatments, cases of many years' standing eight to twelve applications. Before resorting to surgical intervention a trial with ultrasound is recommended. According to Meyer[16] ultrasound is excellently suited for the treatment of angina of the neck accompanied by painful swallowing, but fails in chronic maxillary sinusitis as well as in parotitis. Schwartz[25] noted excellent improvement in treatment of nonallergic sinusitis. Aldes and his group[1] reported very favorable results from ultrasonic treatment of herpes zoster and postherpetic neuralgia involving the ophthalmic branch of the trigeminal nerve, the skin of the face and of the neck.

Undesirable side effects, however, were more likely as long as only sound heads with spot high intensity concentration were available. Pfander and Theismann,[21] for instance, encountered marked attacks of cough when lateral or medial regions of the neck were subjected to ultrasound in the treatment of scleroderma. Others[24] came to the conclusion that ultrasound is only marginally effective, and ascribed most of the surprising successes to the power of suggestion. Still more important, certain hazards of the central beam technique were experimentally demonstrated. Neumann[18] showed in animals that the destructive doses required for treatment of otosclerosis and diseases of the inner ear are equally dangerous when applied in the maxillary antrum, over the cranium, or in the area of the trigeminal nerve. Doses of up to four watts, for a period of up to 15 minutes at a time, and administered by the central beam technique and movement of the sound head, would of necessity be damaging, somewhat on the order of using a sledge hammer rather than gentle finger tip for massage.

As stated before, earlier evaluations, as for instance those by Krusen and his group,[14,19] were based exclusively on experiences with

the one-crystal central beam sound head using high potentially destructive wattages.

The small animals used were anesthetized, otherwise the animals would move due to pain. Since that time, however, decisive progress has been made in the technique of ultrasound therapy. Formerly the applicator consisted of a single crystal head with a high central beam, and the fact that it had to be constantly moved made for unreliable calibration.[22] There is no question that the single crystal moving sound head technique when in good hands, and under careful supervision can produce good results in ultrasound therapy. However, constant careful movement and continuous supervision by a highly trained therapist is necessary. This technical difficulty is avoided by the use of a multiple crystal head without spot concentration, which can remain stationary over the treatment area without the danger of producing tissue damage. Also, this large area technique gives more certainty of covering the site in needful ultrasound. The idea of a multiple crystal sound head was first described by Hintzelmann[19] and by Nencki,[20] who stressed that the flexible "sound cushion" utilizes sonations of minimal wattage, thereby eliminating the danger of overdosage. A further step was taken by Zach,[31] who introduced harmonically tuned multiple crystal sound heads with five to seven quarts crystals.[30]

By this technique there results a sound field without spot concentration, and of a uniform pattern, so that a constant dosage can be applied, regardless of the area of contact. Thus the sound head can remain stationary for applications of from 10 to 16 minutes depending on the wattage per square centimeter used, and the type of tissue being sonated. It is an important consideration that various tissues of the body have different densities, and therefore vary in their ability to transmit ultrasonic waves. When these sonic waves reach a tissue of greater density a relative thermal reaction is created, and heat, though very slight, is produced. Continuous personal attention to the machine and the treatment is no longer required. The instrument switches itself off automatically at a pre-set time. A feeling of warmth of the sound head is occasionally experienced, especially over sensitive areas, but it should be borne in mind that the heat in the sound head is not necessary for proper ultrasound therapy. Zach[29] has successfully employed this new stationary technique in many thousands of cases, nearly 4000 treatments last year. In the meantime a multiple crystal sound head, measuring 22 square centimeters in contact area has become commercially available. All of these recent technical advances seemed sufficiently impressive to warrant

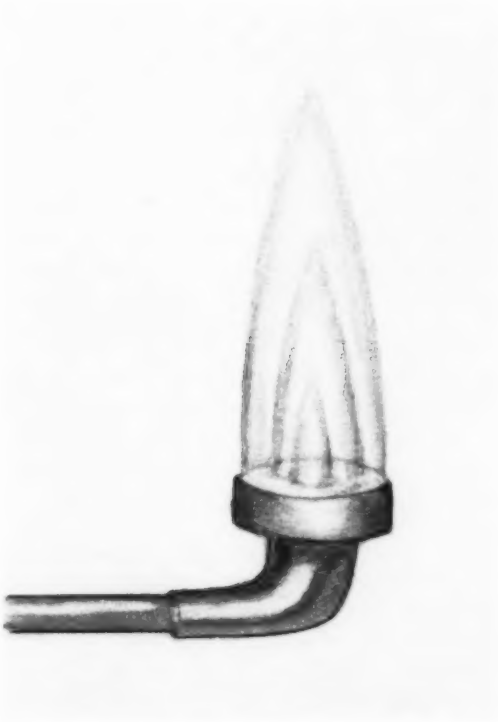

Fig. 1.—Energy pattern of a single crystal sound head (strong center beam).

a new trial of ultrasound in the treatment of various ear, nose and throat diseases. Within the last years several hundred ultrasound treatments have been given, including 130 cases of temporomandibular joint syndrome, temporal neuralgia, acute and chronic otitis externa, acute and chronic frontal and maxillary sinusitis, acute and chronic sore throat, various types of paresthesia and neuralgia, as well as keloids and complications of scar tissue formation. The results were as follows:

Improved	97
Unimproved	21
Worse	2
Unknown	10

In addition, ultrasound has been used in many cases for one or two applications, and discontinued because it became evident that this particular type of treatment was no longer needed or indicated, the patient having recovered.

Of the two cases listed as getting worse with treatment, one was diagnosed after three treatments as a brain tumor. The other, initially thought to be bilateral stellate ganglion neuralgia[27] was subsequently recognized as whooping cough, and this seventy-eight year old woman had to be hospitalized. Of the cases listed as unimproved, some received only a few ultrasound treatments, and it is entirely possible that with continued therapy they might also have responded. The same applies to those patients who for a variety of reasons did not return for therapy. As rigorous clinical standards were applied in evaluating the success of therapy, we may safely state that in a great majority of cases ultrasound is a valuable adjunct to other treatment methods. In many cases it was highly successful as the only treatments given. This was especially so in cases of acute and chronic pharyngitis, tonsillitis, lingual tonsillitis, temporomandibular joint syndrome and various neuralgias, tics and paresthesias.

A careful record of each case was kept, noting the area sonated, the objective, the wattage applied, as well as the duration of exposure at every treatment session. Other types of therapy used in conjunction with ultrasound were also entered, together with the subjective and objective results obtained. Such a combined treatment plan introduces, of course, a variable factor. Yet, our records indicate that with the addition of ultrasound to the standard methods of antibiotic and other systemic or local therapy, recovery was much faster and recurrences were less frequently observed. Thus it may be concluded that ultrasound contributed greatly to the success of such combined therapy.

Ultrasound is excellently suited for prompt relief of pain due to acute inflammation. There was never any complaint of pain due to treatment, even when ultrasound was applied over bony promontories. One patient stated that pain was increased when the cold sound head was brought in contact with the injured temporomandibular joint, but discomfort was no longer experienced when the sound head was pre-heated before applying it to the treatment area. In two or three of the earlier cases on whom a larger sound head was used, patients reported a sensation of moderate heat. Nothing similar has happened since the change was made to a sound head with a total effective diameter of 22 square centimeters. Another advantage of

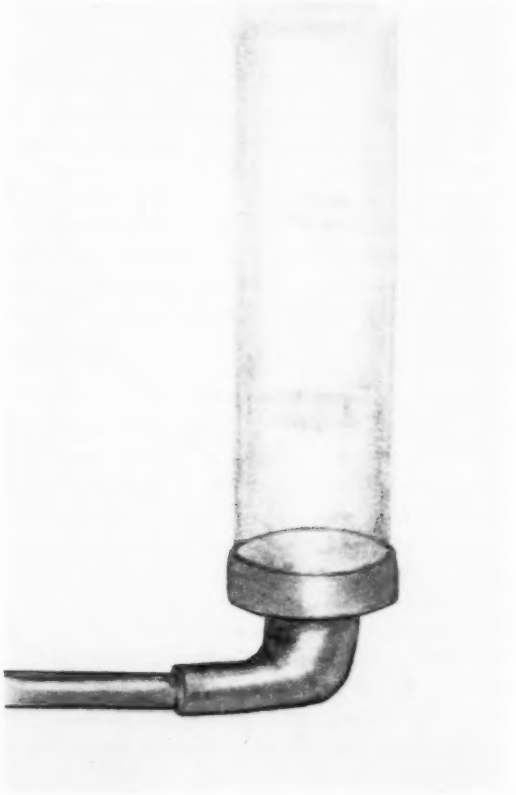

Fig. 2.—Energy pattern of a multiple crystal sound head (uniform beam without spot concentration).

the new sound head is that it fits better onto the shape and angular curvatures of the head and neck. The patient can keep the sound head safely and accurately in place with his own hand. When sonations are applied to the back of the neck, the sound head is fixed with a rubber strap to the treatment area, and held in place in front by the patient.

The optimal dose was worked out by experimentation, in consultation with technical experts. Thus it was established that a maximum dose of 0.25 watts per square centimeter, pulsed plane wave, uniform

energy pattern, and a peak of 0.7 watts per square centimeter, applied for a period of 10 minutes was most effective therapeutically. Acutely inflamed tissue is more sensitive, and in some cases the dose may be somewhat reduced. In this series of cases, however, dosage reduction did not prove necessary, and no discomfort or undesirable side effects were noted. Exact timing is not a problem because the apparatus is fully automatic and cuts off after a pre-set number of minutes. It is well known that ultrasound will not pass through air. In order to bring the sound head in direct contact with the area to be treated, and to eliminate air pockets or bubbles and other interferences, Pond's liquefying cream was used as a coupling agent. Sonations are given with the patient sitting in a treatment chair. A special treatment room is not required. My own ultrasound equipment rests on a small shelf on the wall.

COMMENT

Ultrasound therapy, using a multiple crystal sound head, has proven very satisfactory in the treatment of many ear, nose and throat, head and neck conditions. It is our belief that ultrasound produces "micro-massage" on the cellular as well as on the intracellular level. This results probably in an effect on intracellular metabolism, through alteration of membrane diffusion or osmosis. The favorable response of keloids and various scar tissues is not surprising, as similar successes have been reported by others.[15] A further reason for the good results obtained with the multiple crystal sound head is that sonations can be strictly confined to the involved area, and the applicator remains stationary. It has been demonstrated in animal experiments that damage to the eyes is due to the high concentrations of the central beam directed specifically at the eyes.[4] The multiple crystal sound head, using very low wattage, eliminates any dangerous central beam and injury to the eyes or other tissues. Likewise, injury to the brain could follow only central beam therapy, so abnormally used as to be directed specifically at the organ with high intensity over a prolonged period of time. Even then only a small fraction of the ultrasonic rays would reach the brain tissue itself due to the sonic wave transmission variance of the bony skull. No untoward effects were observed in my cases following my previously described multiple crystal sound head technique where sonations were given over the frontal sinuses, even in the acute stages, including osteomyelitis of the frontal bone, in close proximity to the eyes and brain, or over distributions of the trigeminal nerve.

It is realized that clinical results in a series of patients in which ultrasound was usually combined with conventional local and systemic

therapy, permit no final conclusions as to the ultimate value of sonations for various ear, nose and throat conditions. For that purpose it would be necessary to restrict the treatment to ultrasound, and in addition run a control series in which patients believe that they are receiving ultrasound while in reality the machine is not turned on. Yet, the combinations of ultrasonic therapy with other modalities may have advantages of its own. Behrend and Weiss[5] have shown that in the management of other diseases combined therapy resulted in a striking acceleration of the recovery rate. The good results in the present series may furthermore be partially due to the fact that as a routine attention was given to supplementation of dietary deficiencies,[6] and various stress factors, based on the findings of Selye,[26] and Watson.[28] As demonstrated by Kasahara and Kawashime,[12] ultrasonic waves result after a few seconds or minutes in destruction of a considerable amount of Vitamin C, and it is entirely possible that these and similar undesirable side effects of ultrasound can be balanced by concomitant diatotherapy. Due to recent technical advances and possibly also the combination with other methods, ultrasound is now proving itself to be a valuable adjunct in the treatment of various ear, nose and throat conditions. In the near future we plan to report on experiences in a larger series of patients, including cases treated only with ultrasound, and a sufficient number of controls, carried out in a large charity hospital.

SUMMARY

Ultrasonic therapy, using a twenty-two square centimeter multiple crystal sound head, and very low wattage of 0.25 watts per square centimeter with uniform intensity at less than maximum tissue tolerance has proven a valuable adjunct in the treatment of various ear, nose and throat, head and neck conditions, including acute and chronic sinusitis, otitis externa, acute and chronic pharyngitis, tonsillitis, temporomandibular joint syndrome and other neuralgias. Experiences in a series of one hundred thirty cases are reported and the technique of ultrasound application is discussed. The literature on the subject is briefly reviewed.

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REFERENCES

1. Aldes, J. H., Edmundson, F. B., Agresti, M. L., and Rhoden, G. V.: The Use of Ultrasonic Radiation for the Treatment of Herpes Zoster. Proc. 4th Conf. Ultrasonic Therap., pp. 115-123, 1955.

2. Altmann, F., and Waltner, J. G.: The Treatment of Ménière's Disease with Ultrasonic Waves. A.M.A. Arch. Otolaryng. 69:7-12 (Jan.) 1959.

3. Altmann, F., and Waltner, J. G.: The Treatment of Ménière's Disease with Ultrasonic Waves. Annals of Otology, Rhinology and Laryngology 68:750 (Sept.) 1959.

4. Baum, G.: The Effect of Ultrasonic Radiation. Am. J. Ophth. in Excised Animal Eyes - Preliminary Report 42:696-706 (Nov.) 1956.

5. Behrend, H. J., and Weiss, J.: Ultrasonics in Combined Therapy. Proc. 3rd Conf. Ultrasonic Therap., pp. 110-113, 1954.

6. Currier, W. D.: Combined Nutritional and Psychosomatic Management of Mucosal Disease. Laryngoscope 68:1819-1826 (Oct.) 1958.

7. Denier, A.: Die Ultraschall Therapie, in: Der Ultraschall in der Medizin. Zurich, S. Hirzel, pp. 240-243, 1949.

8. Edmundson, F. B.: Symposium on Ultrasonics; Ultrasonic Therapy in New Zealand. Am. J. Phys., Med. 33:41-44 (Feb.) 1954.

9. Edmundson, F. B.: Treatment of Sinusitis by Ultrasonic Therapy. Proc. 4th Conf. Ultrasonic Therap., pp. 109-114, 1955.

10. Hintzelmann, U.: Ultraschall in der inneren Medizin. Ultraschall in Med. 7:183-185 (Dec.) 1954.

11. Ironside, Wm. M. S., and Lindsay, J. R.: Ultrasonic Therapy for Relief of Vertigo Due to Ménière's Disease. Laryngoscope 69:7:899-913 (July) 1959.

12. Kasahara, M., and Kawashima, K.: Über den Einfluss der ultraakustischen Wellen auf das Vitamin C. Klin. Wochenschr. 16:1191 (Aug. 21) 1937.

13. Krejci, F.: Klinische und experimentelle Erfahrungen über dis Behandlung der Trigeminusneuralgie mit Ultraschall. Wein Klin. Wochenschr. 63:241-242 (Mar. 30) 1951.

14. Krusen, F. H., Herrick, J. F., Schlotthauer, C. F., Wakim, K. G., Bennett, W. A., Janes, J. M., Lambert, E. H., Nelson, P. A., Anderson, T. P., De Forrest, R. E., and Treanor, W. J.: Present Status of Use of Ultrasonic Energy in Physical Medicine. J.A.M.A. 148:646-651 (Feb. 23) 1952.

15. Kuitert, J. H., Davis, M. J., Brittis, A. L., and Aldes, J. H.: Control of Keloid Growth with Ultrasonic Energy. Proc. 3rd Conf. Ultrasonic Therap., pp. 120-125, 1954.

16. Meyer, E.: Ultraschallbehandlung in der Hals- Nasen- Chrenheil- kunde. HNO 2:52-54 (Apr.) 1950.

17. Mülwert, H., and Voss, O.: Eine neue physikalische Behandlungs-methode chronischer Schwerhörigkeit und deren Ergebnisse. Arch. Chren- Nasen- Kehlkopf-heilkunde 119:81-115 (Sept.) 1928.

18. Naumann, H.: Ultraschall und Chr-Region. Arch. Chren- Nasen- Kehl-kopfheilkunde 160:240-292 (Nov.) 1951.

19. Nelson, P. A., Herrick, J. F., and Krusen, F. H.: Ultrasonics in Medicine. Arch. Phys. Med. 31:6-19 (Jan.) 1950.

20. Nencki, L.: Über therapeutische Erfahrungen mit einem neuem Gerat zur statischen Mehrfachbeschallung (Schwingkissen). Ultraschall in Med. 7:191-192 (Dec.) 1954.

21. Pfander, F., and Theismann, H.: Über das Auftreten von Reizhusten bei der Anwendung von Ultraschall im Halsgebiet. Strahlentherap. 80:313-316, 1949.

22. Phillips, K., Smith, E. M., Biro, L. P., Boynton, B. L., and Zach, F. S.: Technique and Clinical Application of Ultrasound Energy. Brit. J. Phys. Med. 17:103-109 (May) 1954.

23. Rosenthal, H.: Ultrasonics in Clinical Medicine. J. Aviation Med. 21:265-272 (June) 1950.

24. Schmalix, J.: Praktische Bedeutung des Ultra-Schalles für die Oto-Rhinologie. Med. Klin. 45:564-565 (May 5) 1950.

25. Schwartz, F. F.: Ultrasonics in Medicine. Brit. J. Phys. Med. 20:179-181 (Aug.) 1957.

26. Selye, H.: The Stress of Life. New York, McGraw-Hill Book Company, 1956.

27. Wallace, L., and Zach, F. S.: Ultrasound—Its Effect on the Cardiovascular System When Applied over the Carotid Sinus and Stellate Ganglion. California Medicine 91:21-23 (July) 1959.

28. Watson, G.: Vitamin Deficiencies in Mental Illness. J. Psychol. 43:47-63 (Jan.) 1957.

29. Zach, F. S.: Clinical Considerations of Static Ultrasound Therapy. In press.

30. Zach, F. S., Boynton, B., Phillips, K., and Smith, E.: Localized Application of Ultrasonic Energy. Brit. J. Phys. Med. 20:175-179 (Aug.) 1957.

31. Zach, F. S., Hausler, H. P., and Lindgren, D. W.: U. S. Biology and Med. 1 (Feb.) 1956.

LXXVIII

THE STERNOMASTOID MUSCLE APPROACH TO PAROTID GLAND TUMORS

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The frequent recurrence of mixed parotid gland tumors following the use of the enucleation technique has made this procedure unacceptable.

Using the enucleation technique recurrence rates from 20-45 per cent are reported by Foster,[1] Ahlbom,[2] McFarland,[3] and Frazell.[4] Using superficial lobectomy or total parotidectomy, Frazell was able to lower his recurrence incidence to 4.8 per cent and Bailey[5] reported no recurrences in a 10 year follow-up.

Needle biopsy and open biopsy are procedures condemned by this author as well as by many others. Permanent partial paralysis has resulted after an open biopsy and there also have been cases where inadequate biopsy confused the diagnosis and delayed a definitive procedure.

Bailey[5] popularized McWhorter's[6] concept that the parotid gland was formed by a deep and superficial lobe connected by an isthmus, with the facial nerve lying between the lobes and dividing into its temporofacial and cervicofacial division at the isthmus. It makes little difference if the gland is divided into lobes, the important point being that the glandular tissue can be separated from the facial nerve without injury to the nerve.

Mobilization of the gland and exposure of the facial nerve are necessary for adequate excision of most parotid tumors. If the tumor lies in the deep lobe, then superficial lobectomy makes the tumor accessible. Occasionally a mixed tumor will be "dumbbell"-shaped and lie in both deep and superficial lobes. This has occurred twice in the author's experience, and was handled by retracting the lower branches of the nerve over the top of the superficial portion of the

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tumor. On only one occasion has the author found it necessary to sacrifice a main branch of the nerve, and this was a case in which the mandibular division was involved by an infiltrating squamous cell carcinoma. The principles about to be enumerated have been found effective by the author in a variety of conditions other than mixed tumors. These include tuberculosis of the gland, chronic mycotic infection, suppurative inflammatory conditions, cysts and malignant tumors.

THE STERNOMASTOID MUSCLE APPROACH

1. Adequate elevation of skin over the parotid and over the mastoid and sternomastoid areas:

This is accomplished most adequately through an incision beginning close to the back of the ear, a little below Henle's spine and carried down over the mastoid to the angle of the jaw. Then it is carried anteriorly below the horizontal ramus of the mandible. An extension of the incision anterior to the ear is optional.

2. Separation of the fascia attaching the anterior border of the sternomastoid muscle to the parotid:

This important maneuver exposes the facial nerve which is found 8 mm deep to the tympanomastoid fissure.[7] Violation of the capsule of the parotid will injure the cervical branch of the nerve, at the same time entering the muscle belly posteriorly is an unnecessary detour.

3. Separation of the parotid gland from the external auditory canal in the almost bloodless plane between the canal and gland:

At the lower end of this dissection dense fibers hold the gland to the tympanic portion of the mastoid and the styloid process. One should use the electrical stimulator in this area before these fibers are cut.

4. Superficial lobectomy:

This is accomplished using blunt and sharp dissection to separate the gland from the facial nerve. If the tumor lies in the deep lobe it may be exposed and removed while the nerve is retracted.

SUMMARY

Superficial lobectomy and total parotidectomy are the procedures of choice in dealing with tumors of the parotid gland.

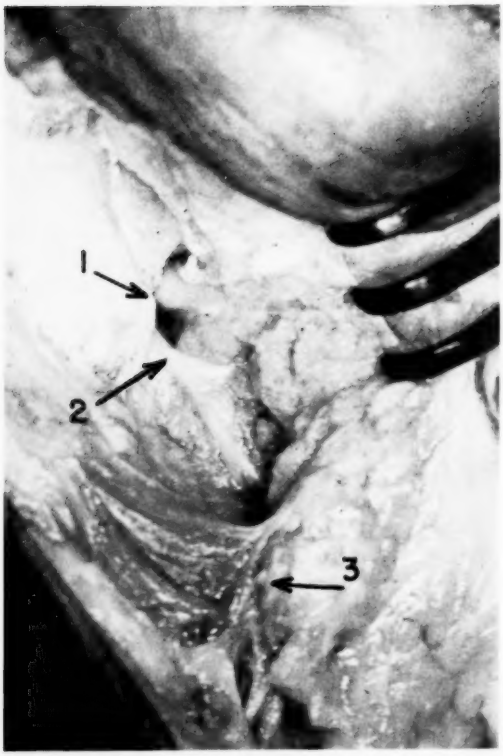

Fig. 1.—Photograph illustrating the sternomastoid muscle approach to the facial nerve. The right ear is retracted upward and anteriorly, showing: 1) the tympanomastoid fissure; 2) the facial nerve continuing in the same line as the tympanomastoid fissure; 3) attachments of the parotid fascia to the sternomastoid muscle.

A simple method to accomplish the above procedures without injury to the facial nerve is presented.

300 East Third St.

REFERENCES

1. Foster, R. P., Tucker, A. L., and Chamberlin, J. A.: Surgical Treatment of Parotid Tumors: Presentation of Current Approach and Evaluation 50 Cases. Am. J. Surg. 24:927-938, 1958.

2. Ahlbom, H. E.: Mucous and Salivary Gland Tumors: Clinical Study with Special Reference to Radiotherapy, Based on 254 Cases Treated at the Radiumhemmet, Stockholm. Acta Radiol. Supp. 23:1, 1935.

3. McFarland, J.: Mysterious Mixed Tumors of the Salivary Glands. Surg. Gynec. and Obst. 76:23, 1943.

4. Frazell, E. L.: Clinical Aspects of Tumors of the Major Salivary Glands. Cancer 7:637, 1954.

5. Bailey, H.: The Surgical Anatomy of the Parotid Gland. Brit. M. J. 2:245, 1948.

6. McWhorter, G. L.: Relations of Superficial and Deep Lobes of the Parotid Gland to Duct and Facial Nerve. Anat. Rec. 12:149, 1917.

7. Kratz, R. C., and Hogg, S.: Surgical Exposure of the Facial Nerve. J. Laryng. and Otol. 72:902-905, 1958.

LXXIX

THE ASSOCIATION OF NERVE DEAFNESS AND RETINITIS PIGMENTOSA

INTERVAL REPORT

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In 1944 I reported the audiometric and vestibular findings on four brothers with nerve deafness and retinitis pigmentosa.[1] Since that time a number of interesting studies have appeared in the literature describing additional cases of hereditary nerve deafness, not only when associated with retinitis pigmentosa, but also with renal disease, goiter, obesity and diabetes, and neurologic disease.

Recently I have had the opportunity of re-examining the four brothers. The purpose of this paper is to report the condition of their eyes and ears 20 years after the initial examination, and to ascertain, in so far as possible, the present status of the children and grandchildren of the four boys and their normal four brothers and one sister.

The subject of familial deafness is not without practical importance, particularly in view of the current advances in distinguishing between inherited and acquired deafness. The incidence of hereditary deafness is difficult to determine. Van Egmond[2] states that, "The number of cases of congenital deafness by heredity is presumably half to one-third of all the deaf, recessive deafness strongly predominating." In a recent survey[3] of a large group of nerve-deaf children, hereditary deafness comprised about five per cent of the group, although in this study the cause was undetermined in 45 per cent of the children. Johnsen[4] found 40 per cent of his cases of high tone perceptive deafness in children to be of genetic origin, and constitutes, according to this author, the commonest cause of perceptive deafness in children. Other investigators[5] have found the hereditary factors in cases of congenital deafness to vary from four to 15 per cent. Increased knowledge of the forms of acquired perceptive deafness secondary to erythoblastosis fetalis, maternal rubella, birth trauma,

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and the ototoxic drugs have tended to decrease the number of cases ascribed to hereditary.

The role of the otologist is to identify the hereditary nature of the condition, to differentiate the cases of familial perceptive deafness from familial conductive deafness, advise the patient as to its genetic implications and to caution him on the futility of medical and surgical treatment. As Cawthorne[5] has remarked, many of these patients now hopefully apply as candidates for the surgical correction of their deafness.

The complexity of the problem of hereditary deafness can readily be seen in a consideration of Cawthorne's[5] classification which is more inclusive than the one proposed by Van Egmond who divides hereditary deafness into two types, dominant inner ear deafness developing at middle age and progressive in character, and recessive sporadic deafness. In Cawthorne's classification, the cases of nerve deafness I reported in 1942 are catalogued under the abiotrophies, associated with retinitis pigmentosa of the recessive type. On the basis of the audiograms to be presented today it would appear this family belongs under the heading of the aplasias, congenital and nonprogressive deafness, in which a localized aplasia of the basal turn of the cochlear duct produced a high tone perceptive deafness. The difficulties in classifying familial perceptive deafness, in differentiating clinically and histologically between acquired and inherited nerve deafness, in establishing the probable hereditary pattern, and in recognizing the progressive degenerative familial types of nerve deafness, are well known. The substantial increase in our knowledge in all phases of inherited ear disease, especially in the last two decades, warrants our continued attention.

The interest in the association of nerve deafness in cases of retinitis pigmentosa has been encouraged by the precise evaluation of the degree of hearing impairment made possible by modern audiometric methods. The incidence of this association is not known, and has been variously estimated between two and 43 per cent.[6] Fowler and Basek[7] found one child, in their series of 81 children under ten years of age with deafness of prenatal origin, to have retinitis pigmentosa. Bell stated that more than 10 per cent of cases of retinitis pigmentosa have nerve deafness. Cawthorne[5] says, "In pedigrees where deafness and this pigmentary degeneration are associated, about 75 per cent of those showing retinal changes also have hearing defects."

Landau and Feinmesser[6] presented the results of the audiometric and vestibular examinations in 22 patients with retinitis pigmentosa.

CAWTHORNE'S CLASSIFICATION OF FAMILIAL PERCEPTIVE DEAFNESSES

I. APLASIAS

CONGENITAL AND NON-PROGRESSIVE DEAFNESS

1) Pure hearing defects. In decreasing severity
   1. Michel (1863) type of "Deaf-Mutism"—Total Deafness
   2. Mondini (1791) type of "Deaf-Mutism"—Total Deafness
   3. Cochleo-succular arrest (Scheibe, 1892)—Subtotal or Partial Deafness
   4. High Tone Perceptive Deafness (Alexander, 1927)

2) Hearing defect in association with other abnormalities
   1. Congenital Meatal Atresia
   2. Retinitis Pigmentosa
   3. Anomalous Pigmentation and Mongoloid appearance (Mende, 1926)
   4. Lateral displacement of inner canthi, a broad nose root (both almost invariable), different colored eyes and white forelock (Fisch and Renwick, 1956)

II. ABIOTROPHIES—HEREDODEGENERATIVE NERVE DEAFNESS

ACQUIRED AND PROGRESSIVE DEAFNESS

1) Pure hearing defects—Innenohrschwerhörigkeit (Albrecht, 1925, 1926)
   1. Due to dominant inheritance (Albrecht, 1923; Johnsen, 1952)
   2. Due to recessive inheritance (Ford, 1952)
   3. Due to sex-linked recessive inheritance (Ersner and Saltzman, 1941)

2) Associated with other abnormalities
   1. Retinitis Pigmentosa (Nettleship, 1907-8; 1909), which is of the recessive type
      Sub-types: a) normal vestibular responses (McGovern, 1942)
      b) no vestibular function (Weber, 1940)
   2. Renal Abnormalities
      a) Associated with Guthrie's (1902) Congenital Hereditary Familial Haematuria—Hurst's (1923) Heraditary Familial Congenital Haemorrhagic Nephritis (Alport, 1927)
      b) Associated with Perkoff et al.'s (1951) Hereditary Interstitial Pyelonephritis (Dolowitz and Stephen's, 1952)
   3. Ear-pits and Branchial Fistulae (Fourman and Fourman, 1955)
   4. Congenital Familial Anhidrosis (Helweg-Larsen and Ludwigsen, 1946)

III. DUE TO BLOOD GROUP INCOMPATIBILITIES

— Rhesus Factor (Coquet, 1944; Crabtree and Gerrard, 1950; Goodhill, 1950)

IV. DUE TO FAMILIAL NEUROFIBROMATOSIS

Familial bilateral acoustic neurofibromata (Gardner and Frazier, 1930)

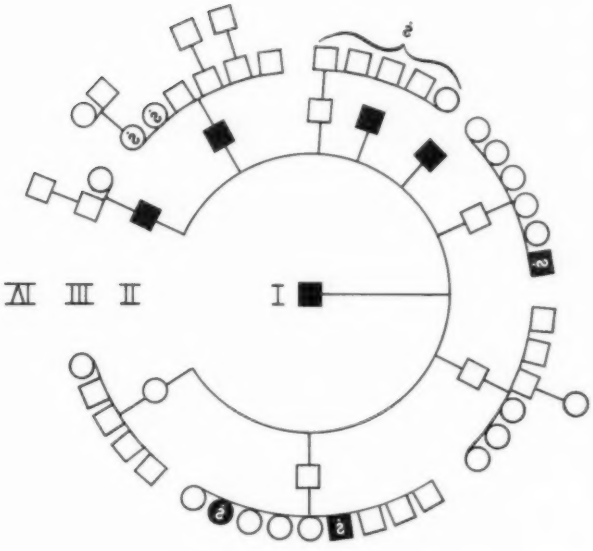

Fig. 1.—Pedigree of the M. family.

Of these, two showed deafness of the conduction type diagnosed otosclerosis; nine patients showed deafness of the perceptive type; one patient was totally deaf; two showed deafness of the mixed type. The vestibular tests were abnormal in four of the 22 patients, all of whom also had impaired hearing. Four of the 22 patients were siblings; 16 of their cases were males and six were females. LaValle[8] studied 12 patients with retinitis pigmentosa audiometrically. All of the patients showed a bilateral symmetrical abnormal audiogram, except for one patient who had had a mastoidectomy. The audiometric pattern appeared to be a low tone perceptive loss. Stenger[9] reported three patients, a brother and sister and an unrelated man, with a recessive type of retinitis pigmentosa, symmetrical nerve deafness and a vestibular abnormality. In 1940 Hardy[10] reported the presence of deafness in three patients with retinitis pigmentosa. Two of the patients were sisters. The audiometric studies showed a symmetrical high tone nerve deafness in all three cases.

An analysis of the clinical otologic findings of the reported cases would indicate a variety of cochlear abnormalities possible when nerve

deafness is associated with retinitis pigmentosa. According to the extent of non-development, the audiometric pattern shows a varying degree of high or low tone perceptive loss, bilateral and symmetrical. The deafness does not appear to be progressive, and the response to the vestibular examination may be normal, decreased, or absent. Reports of histologic examination of the cochlea in these cases are entirely lacking.

The audiometric examination in familial perceptive deafness unassociated with other conditions is characterized by a threshold curve essentially the same in each ear, although a different type of audiogram may be found in various members of the same family. Cawthorne has re-emphasized the fallacy of differentiating types of familial deafness on the basis of the audiogram. The inconsistent audiometric pattern is due to the vagaries of maldevelopment, which, as Cawthorne believes, is according to the degree of aplasia of the inner ear. In separating acquired from inherited deafness, however, Johnsen[11] found the former to have a graph sharply sloping toward the high frequences, whereas the audiogram of the latter tended to be a gradually sloping one. Fisch[12] studied the correlation between the audiometric pattern and many varieties of congenital deafness in 250 children. In his 48 cases of deafness of genetic origin, 11 showed a gently sloping graph, 12 a sharply sloping graph, and 13 showed only a residual hearing for the lower frequencies. In most surveys of this type many cases are classified as of "unknown cause," and Fisch states that heredity is probably often the etiologic factor in this undetermined group. He also shows examples of identical audiograms in two or more members of one family with hereditary deafness, and audiograms in two or more members of one family with hereditary deafness, and audiograms illustrating identical graphs, but in cases of varied etiology (heredity, anoxia, maternal rubella, and hemolytic disease).

When retinitis pigmentosa is combined with sporadic recessive deaf mutism (Usher's syndrome) some histologic observations are available. According to Altmann,[13] the histologic picture is similar to that seen in inherited nerve deafness without retinitis pigmentosa. Nager[14] states there are two principal groups of abnormalities in hereditary deafness, one involving malformations of the osseous and membraneous cochlea, accompanied by nerve degeneration, and the other showing progressive deterioration of the epithelial structures and ganglionic cells of the ductus cochlearis. The first group corresponds with the aphasias in Cawthorne's classification, and the second group, in which the ear develops normally, but a progressive degener-

ative process develops in later life. The latter group include familial premature presbyopia, vulnerability to acoustic trauma, and progressive hereditary nerve deafness either inherited alone or associated with other abnormalities. According to Altmann[13] the pathologic lesion in heredodegenerative deafness is a primary degenerative atrophy of the spiral ganglion and cochlear nerve fibers.

The abnormalities associated with nerve deafness, other than retinitis pigmentosa, have been known for many years, especially in deaf mutism coexisting with mental deficiency, hereditary ataxia, and the combination of deaf mutism, retinitis pigmentosa, mental deficiency and ataxia. Since Cawthorne's report the recent literature records additional cases of nerve deafness associated with other diseases. These cases include the combination of familial deafness and goiter[15]; hereditary renal disease associated with nerve deafness and ocular lesions[16]; retinal degeneration combined with obesity, diabetis mellitus and neurogenous deafness[17]; the familial syndrome of Refsum—retinitis pigmentosa, chronic polyneuritis with progressive paresis of the limbs, ataxia, nystagmus, ichthyosis and neurogenous deafness[18]; the familial association of nerve deafness with nodular goiter and thyroid carcinoma.[19] In the association of familial renal disease and nerve deafness Sturtz and Burke[20] list the criteria necessary for a correct diagnosis; a family history of progressive renal disease in males affected, the presence or history of hematuria, and nerve deafness in the males and in some of the females affected. They state the deafness is a bilateral partial nerve deafness. The deafness is presumed progressive but no audiometric data is available to demonstrate this point. The pathologic factor is not known. The observation of recruitment in one case suggests an end organ type of hearing impairment.

PROGRESS REPORT

In the last 20 years the brothers have been in excellent health. Their vision has progressively deteriorated. In the youngest, J.M., aged 47, the vision has changed from 20/40+3, 20/25—4 to 20/50 in each eye; the next, P.M., aged 56, has changed from 20/200+2, 20/200 to 8/200 in each eye; A.M., aged 58, the second oldest, has changed from 20/40—1, 20/30—2 to counting fingers at two feet in one eye and one foot in the other; the oldest, T.M., aged 62, has changed from 2/200, 5/200 to light perception. The eye findings were characteristic of the lens, retinal and optic nerve changes seen in advancing retinitis pigmentosa.

As illustrated, the audiograms are essentially the same as the ones done 20 years ago. The solid line represents the original audiogram.[1]

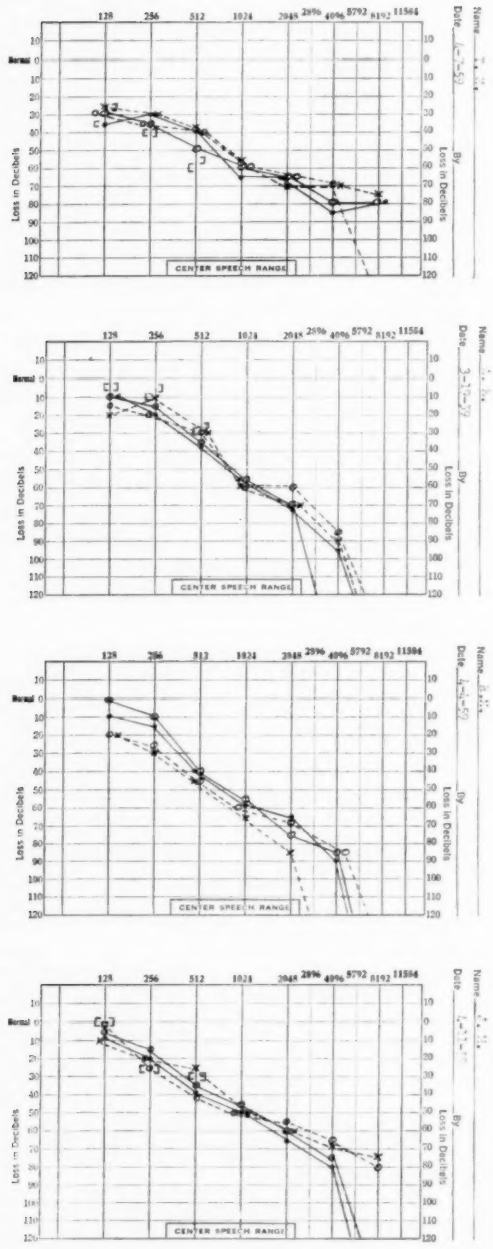

Fig. 2.—Audiograms of the four M. Brothers made in the spring of 1959.

The caloric test repeated on one of the boys showed a normal response on each side.

In the pedigree (Fig. 1) the only members actually known to have the two conditions are the four original brothers; the examination of as many members of the next two generations as possible has not disclosed additional cases. The cases marked with a question mark are those who by history alone are said to have serious eye and ear disease. The family has spread from Virginia to Alaska, and efforts are being made to have local physicians examine these individuals. It has been called to my attention recently the affected boys all have sandy hair and freckles; the rest are brunettes. Some members of the family feel that only offspring with the fair complexion have eye and ear trouble and are considerably concerned when a child of this type is born. This observation is interesting in view of Altmann's[13] comments on the anomalies of the eyes and ears found in merle dogs.

The occurrence of simultaneous familial disease of the eye and ear has provoked considerable discussion among the geneticists, especially in reference to the probability of the combined defects arising from a common hereditary factor. It is generally thought that the involvement of the separate organs are "manifestations of a recessive mutation in a single gene locus." Retinitis pigmentosa alone can be transmitted in a dominant, recessive, or sex-linked manner, although Macklin stated years ago that there are eleven types of inheritance in retinitis pigmentosa. The great majority of cases are recessive, although more than one gene, is probably responsible.

Dr. Richard Shaw, geneticist at the University of Virginia Medical School, kindly examined the pedigree here presented. It was his opinion that since retinitis pigmentosa and hereditary nerve deafness are both uncommon, it is unlikely the association is a chance one, and probably a single gene is responsible for both conditions. This may also account for the sandy hair and freckles found in the affected individuals. He thought the mode of inheritance not clear from the pedigree information available, "The great prevalence of boys in the first generation and the limitation of the condition to the boys of that sibship is very striking."

The genetic pattern in unassociated familial deafness has been given considerable attention in the literature. Johnsen[21] studied 80 children with high tone perceptive deafness. The deafness was familial in about one-third of the families. The heredity was dominant in

some instances and recessive in other families. He states that partial deafness, transmitted by recessive inheritance, is not progressive in type, whereas if dominant, the deafness is inclined to show progression. In the pedigrees of progressive hereditary nerve deafness reported by Dolowitz[22] and by Williams,[23] the hearing loss was noticeable after the age of 20 years and was inherited as a dominant trait. The family recorded by Ersner and Saltzman[24] showed a deafness due to a sex-linked recessive inheritance. In Cawthorne's[5] series of heredodegenerative deafness the apparent inheritance was a mixture of dominant, recessive, and sex-linked recessive types. Cawthorne also commented on the significance of the presence or absence of tinnitus, both in differentiating the various kinds of hereditary deafness and in localizing the site of the cochlear lesion. Hereditary unilateral deafness is extremely rare. The transmission of this condition appears to be a dominant one with low penetrance.[25]

SUMMARY

A twenty-year progress report of four brothers with the association of nerve deafness and retinitis pigmentosa is presented. The audiometric survey showed little change in the type and amount of hearing loss. The vision showed the deterioration characteristic of the course of retinitis pigmentosa. A review of the pertinent current literature is also recorded.

139 S. Main St.

REFERENCES

1. McGovern, F. H.: Association of Nerve Deafness and Retinitis Pigmentosa. Arch. Otolaryngol. 36:827-836 (Dec.) 1942.

2. Zonderman, B.: Preschool Nerve-Deaf Child: Study of Etiologic Factors. Laryngoscope 69:54-59 (Jan.) 1959.

3. Van Egmond, A. A. J.: Congenital Deafness. J. Laryng. and Otol. 68:429-443 (July) 1954.

4. Johnsen, S.: Some Etiologic Aspects of High Tone Perceptive Deafness in Children. Acta Oto-laryng. 44:205-218 (May-June) 1954.

5. Cawthorne, T. E., and Hinchcliffe, C.: Familial Perceptive Deafness. Pract. Oto-Rhin. and Laryngol. 19:69-83, 1957.

6. Landau, J., and Feinmesser, M.: Audiometric and Vestibular Examinations in Retinitis Pigmentosa. Brit. J. of Ophthal. 40:40 (Jan.) 1956.

7. Fowler, E. P., Jr., and Basek, M.: Causes of Deafness in Young Children. A. M. A. Arch. Otolaryng. 59:476-484 (Apr.) 1954.

8. LaValle, R.: Audiogram in Retinitis Pigmentosa. Acta Oto-Rhino-Laryng. Belgica 7:361, 1953.

9. Stenger, H.: Retinitis Pigmentosa, Symmetrical Inner Ear Deafness, Typical Reversible Vascular Fistula Reaction without Fistula and Irritability of Vestibule: Contribution to Combination of Hereditary Eye and Ear Disease. Arch. Ohren-Nasen. u. Kehlkopfh. 170-187-198, 1956. Abstract Year Book E. N. T. 1957-8.

10. Hardy, G.: Deafness and Retinitis Pigmentosa. Am. J. Ophth. 23:315-17 (Mar.) 1940.

11. Johnsen, S.: Hearing Loss of the Perceptive Type. J. Laryng. and Otol. 71:667 (Oct.) 1957.

12. Fisch, L.: The Etiology of Congenital Deafness and Audiometric Patterns. J. of Laryng. and Otol. 69:479 (July) 1955.

13. Altmann, F.: Histologic Picture of Inherited Nerve Deafness in Man and Animals. Arch. Otolaryng. 51:852-890 (June) 1950.

14. Nager, F. R.: Labyrinth Malformations in Light of Present Day Genetics. Pract. Oto-Rhino-Laryng. 13:129-145, 1951.

15. Johnsen, S.: Familial Deafness and Goiter in Persons with Low Serum Level of Protein-Bound Iodine. Acta Oto-Laryng. Supp. 140:168-182, 1958.

16. Goldbloom, R. B., Fraser, F. C., Waugh, D., Aronovitch, M., and Wiglesworth, F. W.: Hereditary Renal Disease Associated with Nerve Deafness and Ocular Lesions. Pediatrics 20:241-252 (Aug.) 1957.

17. Alström, C. H., Hallgren, B., Milsson, L. B., and Asander, H.: Retinal Degeneration Combined with Obesity, Diabetes Mellitus and Neurogenous Deafness. Acta Psychiat. et Neurol. Scandinav. 34:Supp.129, 1959. Abs. 1959-60 Year Book E. N. T.

18. Ashenburst, E. M., Millar, J. H. D., and Milliken, F. G.: Refsum's Syndrome. Brit. Med. J. 415 (Aug. 16) 1958.

19. Elman, D. S.: Familial Association of Nerve Deafness with Nodular Goiter and Thyroid Carcinoma. New Eng. M. J. 259:219-223 (July 31) 1958.

20. Sturtz, G. S., and Burke, E. C.: Hereditary Hematuria, Nephropathy and Deafness. New Eng. Med. J. 254:1123-1126 (June 14) 1956.

21. Johnsen, S.: Hereditary in Perceptive Deafness. Acta Oto-Laryng. 42:539-552 (Dec.) 1952.

22. Dolowitz, A. A., and Stephens, F. E.: Hereditary Progressive Nerve Deafness. Trans. Amer. Acad. Ophth. Otolaryng. 56:457, 1952.

23. Williams, F., Lyons, J. P., Stein, S. P., and Roblee, L.: Progressive Hereditary Nerve Deafness: Report of Four Cases in One Family. M. Ann. Dist. of Columbia 23:681-683 (Dec.) 1954.

24. Ersner, M. S., and Saltzman, M.: Progressive Analogous Nerve Deafness in Three Successive Generations with Sex-limited Inheritance. Laryngoscope 51:241-45 (Mar.) 1941.

25. Everberg, G.: Hereditary Unilateral Deafness. Acta Oto-Laryng. 47:303 (Apr.) 1957.

LXXX

SUBLINGUAL SWELLINGS

A NEW APPROACH

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Surgery of the floor of the mouth deals mainly with ranula, dermoid cysts, lesions of Wharton's ducts, lesions of the sublingual glands and exposure of the lingual nerve.

Conditions needing surgical treatment are operated on by the general surgeon, with the patient in the half-sitting position. Though the cysts in this area are small, yet the difficulty encountered in this position has been stressed in many textbooks of surgery.

As for *ranula,* Hamilton Bailey[1] expressed the difficulty of complete removal, and many others, meeting with this difficulty, went so far as to say that complete enucleation is impossible. Many surgeons are not satisfied to remove as much as possible of the cyst wall and the cut edge of the cyst wall is then sutured to the edge of the mucous membrane, i.e., partial excision with marsupialization. For the plunging ranula, it is said that it *must* be approached through the neck[1]; he added that sometimes by this route complete extirpation is possible, but occasionally complete removal is impossible.

Sublingual dermoid cysts are divided into median and lateral. Each is again divided into those situated above and those situated below the buccal diaphragm, the mylohyoid muscles. Most of them are above the mylohyoid muscle.

Treatment of all varieties of sublingual dermoid cysts is carried out by dissection through an external incision beneath the mandible.[1,2]

The case described below has two loculi, one presenting just under the mucous membrane of the mouth and the other in the submental region, with an isthmus connecting both loculi. For this type I use the term "plunging" dermoid cyst, as for a plunging ranula.

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Calculus in the submaxillary duct is frequently operated on with the patient in the half-sitting position. Removal is preceded by measures to prevent the stone from slipping back towards the hilum of the gland, either by clamping the duct or by passing a stitch under traction to kink the duct.

The chief cause of the difficulty encountered by the general surgeon is that he operates with the patient in the semi-sitting position. The disadvantages of this position are many: first, that the tongue overlies the mass and a continuous elevation of the tongue by an assistant is necessary. Ideally the tongue should be pulled up, back and to one side, which is not easy with the patient thus seated.

The next difficulty is that blood tends to collect in the most dependent parts of the field, which is the area where the surgeon is working. Frequent clearing of the field by suction or mopping is thus required, and the time of the operation is nearly doubled by so doing. Thirdly, there is the major problem of keeping the field of operation well illuminated. The surgeon stands by the right side of the patient, light being on the left side, directed from behind and above the surgeon's right shoulder, and the hand throws a shadow over the field of operation. Also the assistant who is pulling the tongue or using continuous suction interferes with proper illumination. Obviously in this position of patient and surgeon it is impossible to illuminate the area behind the symphysis menti.

The present approach of the sublingual region is free from all above mentioned disadvantages. The approach is so convenient and illumination so satisfactory that it was possible to dissect between the two genioglossi and the two geniohyoids and further to go through the diaphragma oris to the submental region as will be shown in the following case record.

REPORT OF A CASE

K.Sh., a child 10 years old, had a swelling under the tongue and another in the submental region, dating back to infancy. Two years ago, the swelling behind the chin became very tender and was incised, followed by disappearance of both swellings, but it reappeared a few months later.

Examination revealed a cystic well-defined swelling in the submental region (Fig. 1). The skin showed the scar of the previous incision.

Figure 1

Figure 2

Figure 3

Examination from the inside of the mouth revealed a rounded swelling under the tongue, in the middle line. The tongue was pushed back and up and could not be protruded (Fig. 2). The ducts of the submaxillary salivary glands and the sublingual glands themselves were stretched over the anterior part of the swelling. Cross fluctation between both loculi could be elicited.

Aspiration required a wide-bore needle as the contents consisted of thick putty-like matter of yellowish white color. Lipiodol was injected into the sublingual portion and x-rays showed trickling of the radio-opaque material to the lower loculus and the constriction between the two loculi is clearly shown in Figure 3.

OPERATION

Anesthesia was administered via an endotracheal tube passed through the nose and the patient's position was exactly as for a tonsil operation. A Davis gag was used, with a special blade which rests only on the lower teeth. The area was infiltrated with saline epinephrin. A transverse incision was made and the cyst wall exposed. Dissection was carried on with small scissors and fine forceps (Fig. 4). The upper loculus was dissected till it began to get narrower towards

Figure 4

the isthmus between the two geniohyoid muscles. The cyst was incised deliberately and evacuated and the lower loculus was explored with a finger. The two geniohyoids were pushed apart and the isthmus followed between the mylohyoid muscles, to the submental region. A finger inside facilitated dissection and every now and then the nurse assisted by gentle pressure over the submental region. Easily the cyst wall was completely removed.

The isthmus was much narrower than is shown in the figure, as it was dilated by a finger put in to feel the extent of the submental loculus.

THE SUBLINGUAL APPROACH IN THE TONSIL POSITION

The advantages of this approach over the operation in the sitting position are that the patient, anesthesia, and surgeon are exactly as for the tonsil operation. A source of light is put on the right side of the table and the operative field is illuminated with reflected light from a head mirror.

A modified Davis gag is used, which rests only on the lower teeth, thus allowing the tongue by its weight to fall towards the palate, in an ideal position to expose the sublingual area without the need of an assistant (Fig. 5).

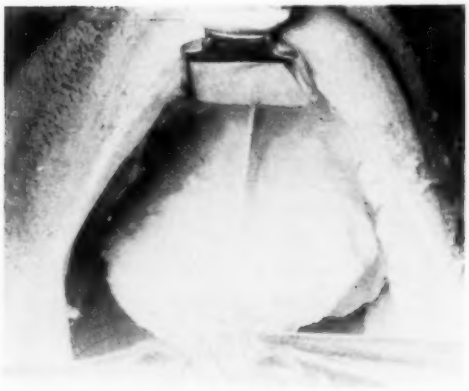

Figure 5

Figure 6

A further advantage is that the area of the operation is at a higher level, so any blood will trickle away from the operative field; thus dissection is carried out always under vision, and no time is lost in suction or mopping the blood.

The incision can be carried either between the tongue and the sublingual papillae or between them and the mandible.

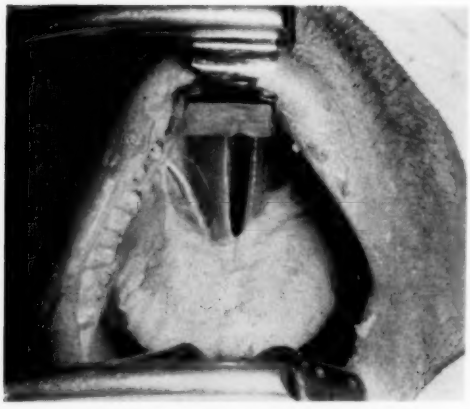

Figure 7

Figure 8

The first is easier to perform, while the second is somewhat difficult as it lies under the shelter of the lower incisors and the blade of the gag. However, the latter incision was used in this case, and was found to give a better access to the submental loculus.

The incision is carried out laterally to allow a convenient exposure of the cyst and the genioglossi but should not be extended too far, as

there lie the lingual nerve and Wharton's duct; their crossing is well shown in Figure 6. It is clear that if the first incision is followed, one will not be free to extend it laterally without endangering these structures, and this is a reason for preferring the second.

The incision can be modified according to the requirements of the operation. It may be done on one side only and extended more laterally if the aim is to remove a stone from the deep part of Wharton's duct. It is evident that the duct can be conveniently exposed as far as the deep part of the submaxillary gland.

Whichever incision is followed, the mucous membrane is dissected to expose the two genioglossi (Fig. 7).

Pushing them apart exposes the geniohyoid muscles and when these are retracted laterally the mylohyoid diaphragm is exposed (Fig. 8).

In the case of the cyst described above, a defect was found in the midline just behind the symphysis menti, and through it the isthmus connected with the submental loculus.

The field of the operation is now deep enough for reflected light from a head mirror to afford the satisfactory illumination. Any blood vessels are ligated over a full curve artery forceps, and the muscles are approximated with one catgut stitch.

In the above case a corrugated rubber drain was inserted, but when the patient recovered he could not tolerate it and it was removed. Some bleeding followed the operation which was controlled by a thick triangular cotton pad in the submental region and tight pressure by a firm bandage passing over the vertex. This was maintained for 48 hours, and no blood or serous fluid collected in the submental region or between the muscles. Antibiotics were given together with antiseptic mouth washes.

Five days after the operation, there was tenderness and some swelling of both submaxillary salivary glands due to an edema, secondary to the operation, which subsided in a week.

SUMMARY

1. A case of "plunging" sublingual dermoid cyst is described.

2. A sublingual approach in the tonsil position was used facilitating complete removal of both upper and lower loculi.

3. The anatomy of the region as viewed from this aspect is described.

4. The advantages of this approach over the classical operation in the sitting position is discussed. This makes it the best approach for ranula and other sublingual cysts, and allows their complete removal without marsupialization.

5. The plunging ranula and dermoid cyst can be removed via this approach and there is no need for an external incision in the submental region or double dissection through the mouth and neck.

6. The approach is indicated for sublingual ranula and dermoid cysts, plunging ranula or dermoid cysts. An excellent view of Wharton's duct is obtained to the deep part of the gland, and this allows removal of a stone at the beginning of the duct, previously an indication for removal of the whole gland. Tongue-tie can be relieved in this position. If there is an indication to section the lingual nerve under vision, it can be exposed by this approach.

KASR EL EINI HOSPITAL

My best thanks to Dr. Hosny Farid for permission to record this case, and for the assistance of Dr. M. Mokhtar and Dr. A. Fatma.

REFERENCES

1. Bailey, Hamilton: A Short Practice of Surgery. Published by H. K. Lewis & Co., London. Tenth edition, pp. 133-136, 1956.

2. Ird, Ian: A Companion to Surgical Studies. E. S. Livingstone Ltd. Second edition, pp. 264 and 314, 1958.

3. Bailey, Hamilton: Pye's Surgical Handicraft. John Wright & Sons Ltd., Bristol. Sixteenth edition, p. 355, 1950.

4. Kinawi, M. M.: Spotlights on Surgery. Published by the Anglo-Egyptian Bookshop, Cairo. First edition, p. 358, 1951.

5. Romanis, W. H. C.: The Science and Practice of Surgery. Published by J. & A. Churchill, Ltd., London. Eighth edition, pp. 217 and 218, 1948.

LXXXI

MIDDLE EAR MUSCLE PROTECTION FROM THE ACOUSTIC TRAUMA OF LOUD CONTINUOUS SOUND

AN ELECTROPHYSIOLOGICAL STUDY IN CATS

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The protective value of the middle-ear-muscle reflex in prevention of acoustic trauma (damage due to loud sound) was hypothesized well over a century ago.[1] The idea seemed logical enough since the tensor tympani and stapedius muscles were seen to contract reflexly when the ear was stimulated with loud sound. Later observations demonstrating attenuation of the cochlear microphonic amplitude during these contractions tended to strengthen this hypothesis.[2] Yet no confirmatory studies were undertaken until recently when a number of investigations have appeared in its support. Usually these experiments have used an impulsive-type noise (gunfire, firecrackers) as the damaging sound. Estimations of the protective value of the middle-ear-muscle reflex have been obtained after exposure by physiological, psychological, or anatomical means.[3-5]

In nature, however, the middle-ear-muscle reflex probably does not protect against sound trauma of this sort. The reflex latency of the muscle contraction is 10 to 15 msec after sound reaches the cochlea. The energy from an impulsive sound would have been dissipated within the cochlea long before this contraction could occur. To circumnavigate this difficulty, both Fowler and Hilding used a conditioning stimulus to produce muscle contraction before the impulse-noise exposure. While this would seem perfectly valid for demonstrating muscle effects, it still seems a good idea to test the effectiveness of the middle ear muscles under circumstances where the trauma-producing sound is itself the contraction-producing stimulus.

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This research was supported by grant No. B-2167 of the National Institutes of Neurological Diseases and Blindness.

A preliminary study of this sort was performed in 1943 but apparently was never completed.[6] In that experiment, behavioral auditory thresholds were obtained on four normal cats. These animals were then exposed to loud (153 db, re: 0.0002 dynes/cm^2) pure tone for one minute. Thresholds obtained six weeks post exposure showed no hearing loss. Then these same cats were anesthetized and their sound exposures repeated at slightly lower sound levels but over longer periods. This time their post-exposure thresholds were markedly elevated. The differences between the two thresholds were attributed either to the effect of the middle ear muscles or to closure of the cats' somewhat tortuous external auditory canal by contraction of the external ear muscles. Possible differences caused by the anesthetic or by the changes in the stimulus itself were not considered.

The experiments to be reported here could be regarded as complementary to the above-mentioned preliminary study. Cats with normal and deactivated middle-ear-muscle reflexes were subjected to a traumatic sound of somewhat longer duration. Post-exposure differences in the degree of cochlear damage were explored and compared electrophysiologically.

METHODS

Eight cats (13 ears) were used in this study. They were divided into three groups as follows: three cats (4 ears) had the insertions of the tensor tympani and stapedius muscles cut; two cats (4 ears) had these muscles deactivated by a general anesthetic (Nembutal)*; four cats (5 ears) had normally functioning muscles. Except as just mentioned, all cats received equal treatment, including the same amount of traumatic sound.

All animals had electrodes permanently implanted on their round windows. Measurement of cochlear potentials obtained through these electrodes, before and after exposure to traumatic sound, served as the comparative index of the degree of damage produced. The use of this experimental animal preparation and the value of cochlear potentials in assessing cochlear damage produced by sound have been de-

* It is well accepted that the middle-ear-muscle reflex is deactivated by barbiturate anesthetic. However, it should be mentioned here that this experimenter has not had occasion to monitor EMG potentials from these muscles in the anesthetized animal at the sound level used here. Such monitoring was not thought necessary since no activity has been seen in previous work up to 110 db (re: 0.0002 dynes/cm^2) and Davis has found no evidence of sound-induced muscle activity in the anesthetized guinea pig at 135 db.[7]

scribed and discussed in detail elsewhere and need not be repeated here.[7,8]

After the surgical procedures mentioned above, each cat was given antibiotics (penicillin and chloramphenicol) and allowed at least ten days for recovery and stabilization. At the end of that time the cat was placed awake in a restraining box. A hearing aid transducer in an ear mould was placed in the external auditory canal and taped in place. Input-output functions for cochlear microphonic in response to tones (1-5 kc) and VIII nerve action potentials in response to clicks were then obtained. If these responses were within normal range, the animal was anesthetized and the measurements repeated so that baseline, muscle-deactivated recordings could be obtained for control purposes. Repeat measurements were made at three- to seven-day intervals after these initial ones to assure stability of responses.

Immediately before traumatic exposure these cochlear potential measurements were again repeated. The cat was then placed in a restraining apparatus which held head movement to a minimum of 1-2 cm in any direction and exposed to a 1 kc tone at 135 db for 2 hours. Following exposure, cochlear potential measurements were repeated within 10-20 minutes and then serially over several weeks so that recovery could be observed. At the end of this period each ear was examined for signs of infection, electrode displacement or other possible artifacts.

The instrumentation used in these experiments was conventional. Tones or condensor discharges were presented in increments of 10 db to the ear under test through the hearing aid transducer (Audivox 9-C). The audio output of the system was kept stable throughout, but no absolute reference level was established. Action potential measurements were obtained directly from the oscilloscope tube face. Cochlear microphonic amplitudes were recorded on a Grass Polygraph after rectification and compression into a logarithmic function by a Mosely Logarithmic Amplifier. The over-all sensitivity of the system was limited to 10 uv when recording from the awake cat.

The traumatic sound was produced in a separate, highly sound-deadened room by a Jensen H-100 driver unit coupled to a DD-100 horn. The sound pressure level was obtained with a General Radio Sound Level Meter and was measured with the cat in position, facing the sound source. Readings were taken in the immediate area of each ear in all its possible positions, and at several times during the sound

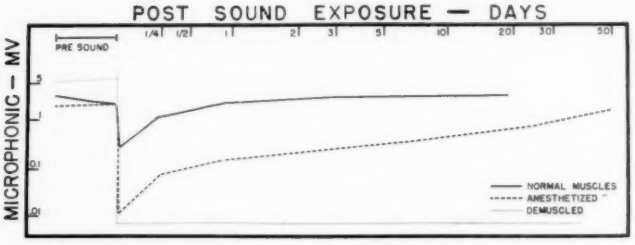

Fig. 1.—Cochlear microphonic amplitude depression with acoustic trauma: Pre- and post-exposure cochlear microphonic responses to a 2 kc tone at about 80 db above 0.0002 dynes/cm^2 are plotted for 3 cats. The animals differ only in the functioning of their middle ear muscles (see code). Ten microvolts was the minimum measurable amplitude.

exposure. Variation in the sound field was less than plus or minus 1½ db.

RESULTS

Figure 1 demonstrates typical depressions in cochlear microphonic occurring with traumatic sound exposure in each type of animal preparation. Pre- and post-exposure amplitude measurements are plotted for a single ear from each group. All showed an initial depression. Note however that the cochlear microphonics from the cat exposed to the tone while awake and with the middle ear muscles active (solid line) returned to pre-exposure levels within 22 hours. In the animal whose middle ear muscles were still intact, but deactivated by anesthesia during exposure (dashed line), the cochlear microphonic was more severely depressed and required about 50 days to return to near normal. In the cat with the middle ear muscles cut (dotted line), damage to cochlear-microphonic-producing structures was even more severe, and there was no evidence of recovery above the 10-uv baseline.

The particular cochlear microphonics plotted in Figure 1 were produced in response to a 2 kc tone. In these experiments, any other frequency between 1 and 5 kc would have demonstrated about the same degree of cochlear microphonic change. For, as mentioned by Davis, the frequency loss recorded at the round window in response to sound trauma is not necessarily specific for the traumatic or any other frequency.

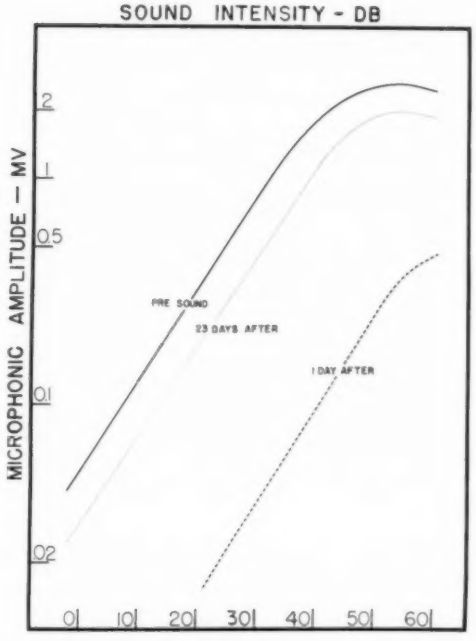

Fig. 2.—Cochlear microphonic response to a 2 kc tone before and after sound trauma: These are round wndow recordings from an anesthetized cat. Decibel values (abscissa) are increments of sound intensity only.

At least above the 10 uv baseline the cochlear microphonic amplitude shift with sound damage is also rather general. The intensity of Figure 1's 2 kc tone was about 80 db above 0.0002 dynes/cm^2. This intensity is near the upper limit of linear response of the cochlear microphonic in the anesthetized cat. Any amplitude of microphonic taken from this linear portion of an input-output function would have shown the same degree of depression. This has also been previously reported.[7] However, as an illustration of these changes in cochlear microphonic, see Figure 2.

Figure 2 is a plot of the input-output functions of the 2-kc cochlear microphonic for the anesthetized cat of Figure 1 over a range of sound intensities. The solid line shows the measurements immediately before acoustic trauma; the dashed line is the cochlear microphonic

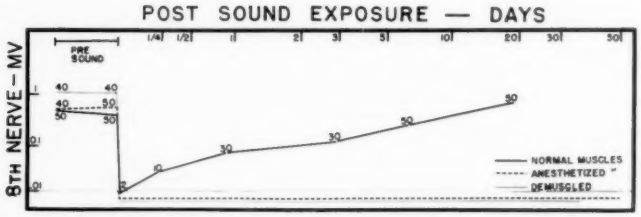

Fig. 3.—Pre- and post-acoustic trauma measurements of the VIII nerve action potential in response to acoustic clicks: The amplitude measured is the maximum peak-to-peak mv of the N1 component not contaminated by cochlear microphonic. The numbers on the plots for 3 ears indicate the decibel range between this peak value and the oscilloscope threshold noise level. The differences in middle ear muscle activities are indicated in the lower right corner.

response over this same sound range at one day post exposure, while the dotted line shows the recovery by the 23rd day.

The post-exposure differences in cochlear integrity among the three groups were even more striking when the measure was the VIII nerve action potential. In Figure 3 these changes with trauma have been plotted for the same three ears as those of Figure 1. The only ear to show any recognizable VIII nerve response following traumatic stimulation was the one with the middle ear muscles intact and active. Complete recovery of amplitude required 21 days. No action potentials ever appeared above the baseline for either of the two types of preparations with the middle ear muscles deactivated.

In Figure 3, the points plotted are expressions of the maximal amplitude of the N_1 component of the action potential measurable without contamination from cochlear microphonic.[9] The numerical values at these points are the decibels of sound between threshold and this maximum value. Threshold was defined as the smallest amplitude of the N_1 component visible above the background noise on the oscilloscope.

Post-traumatic cochlear potential changes in the remainder of the experimental population were essentially those of the three ears used as examples. In all cats the most severe and sustained depressions were seen in the VIII nerve response. But by either of the electrophysiological criteria (cochlear microphonic or action potential), there was

significantly less damage to ears in which the middle-ear-muscle reflex was intact and active. Within this group of 5 muscle-active ears there was variation in the initial degree and eventual duration of potential depressions. Never was the depression so severe or so long lasting, however, as in the other two groups. Of the other two groups, the more severe losses were produced in the surgically demuscled ears, which showed no recovery by either criterion, even after 60 days in one case. The anesthetized cats, although showing no apparent VIII nerve recovery in the time span of the experiments, did evidence some return of cochlear microphonic.

COMMENT

The results set forth above demonstrate that the middle-ear-muscle reflex is effective in the reduction of acoustic trauma when the stimulus is of a non-impulsive variety. The use herein of a two-hour-long traumatic sound shows conclusively that these muscles are capable of sustained protective action. Reflex fatigue, though it may still be a factor, is neither rapid nor complete enough to nullify their effectiveness over this reasonably long period of time.

Though the essential task of this report may be regarded as complete with the foregoing paragraph, there are certain other problems which should be mentioned. Among these is the degree of confidence to be placed in the present electrophysiological measurements as indices of cochlear damage. Although there is general agreement that the correlation of cochlear potentials and anatomical evidence for damage is a positive one, deemed adequate for these experiments, the exact value to be placed on any single measurement of potential is open to question and some dispute.[10,11] This is particularly true regarding cochlear microphonic measurements at the round window. Tasaki and others have shown that there may be local damage along the basilar membrane without major alterations in the round-window cochlear microphonic response. This implies that in order for the round-window-recorded microphonic to show damage, it must be either local in the first half to two-thirds of the basal turn, or quite severe over larger areas further along the cochlear partition. The work of Kobrak substantiates this opinion.[12]

Further consideration of this problem has been facilitated here by the differences between the action potential and cochlear microphonic responses seen in such ears as that of the anesthetized cat of Figures 1 and 3. The microphonic returned to apparent near-normal many days after sound exposure, while the action potential continued

to remain below the 10 uv baseline. Since such an action potential is thought to be recruited over a major portion of the cochlea, its continued depression probably indicates cochlear damage not reflected in the microphonic. Alternative explanations for this behavior do exist. One worthy of consideration is that the action potential is generally more susceptible to acoustic trauma than is the microphonic.

Because of these uncertainties, this author does not feel justified in doing anything more than ranking the degree of damage produced in these expermeints. When both the cochlear microphonic and action potential return to pre-exposure values, as they did in the normally-muscled ears, it is reasonable to assume considerably less cochlear damage than when both electrical indices remained diminished. In the anesthesia-deactivated exposures, where only one of the indices remained markedly depressed, an intermediate degree of damage can be inferred. It is reasonable to assume that this lesser degree of injury may have been due to a damping effect on ossicular chain vibration produced by the inert mass of the muscles themselves. The importance of the general metabolic effects of the anesthetic agent on the cochlea remains for future evaluation.

Also remaining for future consideration are the variables which affect post-injury recovery curves, and the differences noted within the group of cats with normally active middle ear muscles. Since this report represents only a preliminary investigation into these particular problems, additional comments will be deferred until a larger series of animals is available.

SUMMARY

Serial measurements of cochlear microphonic and VIII nerve action potentials were obtained from three groups of cats with electrodes permanently implanted on their round windows. All were exposed to traumatic sound (1 kc for 2 hours at 135 db re: 0.0002 dynes/cm^2). Cats exposed awake and with the middle ear muscles intact and active showed considerably less evidence of trauma that did the cats with either 1) the middle ear muscles deactivated by anesthesia, or 2) both middle ear muscles cut weeks before exposure. Action potential showed more depression than did cochlear microphonic in all cats.

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REFERENCES

1. Müller, J.: Handbuch der Physiologie des Menschen. II, abt. 1, 393-483, 1837; Trans. by W. Baley: Elements of Physiology 744-771, 1843.

2. Hallpike, C. S., Rawdon-Smith, A. F.: The Function of the Tensor Tympani Muscle. J. Physiol., Lon. 81:25-27, 1934.

3. Hilding, D. A.: The Intratympanic Muscle Reflex as a Protective Mechanism Against Loud Impulsive Noise. Annals of Otology, Rhinology and Laryngology 69:51-60, 1960.

4. Fletcher, J. L., Riopelle, A. J.: Effect of a Loud Pre-exposure Tone on TTS Resulting from Impulsive Noise. J. Acoust. Soc. Am. Abstr. 31:831, 1959.

5. Takahashi, H.: Experimental Studies on the Relation Between Sleep on the Acoustic Injury of the Auditory Organ. Hiroshima J. Med. Sci. 3:87-111, 1954.

6. Hawkins, J. E., Lurie, M. H., Davis, H.: Injury of the Inner Ear Produced by Exposure to Loud Tones. Comm. Med. Res., U.S. Off. Sci. Res. and Devel. 1943.

7 Davis, H., et al.: Acoustic Trauma in the Guinea Pig. J. Acoust. Soc. Am. 25:1180-1189, 1953.

8. Simmons, F. B.: Middle Ear Muscle Activity at Moderate Sound Levels. Annals of Otology, Rhinology and Laryngology 68:1126-1144, 1959.

9. Rosenblith, W. A., Rosenzweig, M. R.: Electrical Responses of Acoustic Clicks: Influence of Electrode Location in Cats. J. Acoust. Soc. Am. 23:583-588, 1951.

10. Lawrence, M., Wolsk, D., Burton, R. D.: Stimulation Deafness, Cochlear Patterns, and Significance of Electrical Recording Methods. Annals of Otology, Rhinology and Laryngology 68:5-33, 1959.

11. Tasaki, I., Davis, H., Legouix, J. P.: The Space-Time Pattern of Cochlear Microphonics (Guinea Pig), as Recorded by Differential Electrodes. J. Acoust. Soc. Am. 24:502-519, 1952.

12. Kobrak, H.: The Effects of Limited Cochlear Lesions on Cochlear Potentials and Middle Ear Muscle Reflexes. Am. J. Physiol. (Abstr.) 116:93, 1936.

LXXXII

TECHNIQUE OF HIGH-SPEED PHOTOGRAPHY OF THE LARYNX

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The basic technique of photographing the human vocal cords at speeds of 4000 and more frames per second was developed by the Bell Telephone Laboratories and described in 1940.[1] Their motion pictures were a landmark, clarifying innumerable features of laryngeal physiology and stimulating laryngologists to pursue this fruitful investigative approach. Other films have since been produced, the most outstanding of which are those of von Leden and Moore.[2,3]

In the method developed by the Bell Telephone Laboratories basic equipment consists of a high-speed camera, laryngeal mirror, and powerful light source, all of which are in fixed position relative to each other (Fig. 1). The camera is aligned with the immovable laryngeal mirror through a hole in a much larger mirror. This mirror is set at a 45° angle and reflects light into the mouth. The subject opens his mouth and is guided over the fixed laryngeal mirror until the larynx is visualized in the camera. When the desired view is obtained, the camera is activated by a switch and 100 feet of film are exposed in about one second. The larynx cannot be seen during the actual shooting.

Several years ago the authors undertook an investigation of the human larynx by high-speed photography, following this method. The resulting motion pictures were of excellent quality, but a single

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technical obstacle that could not be overcome in studying the mechanism of vocal registers necessitated the development of an entirely different set-up. Many larynges which can be visualized when a mirror is introduced into the throat cannot be similarly exposed when the subject is brought over a fixed mirror. It requires both practice and patience to adapt a patient to this unusual type of mirror examination in which all adjustments must be made by moving the head rather than the mirror. In an apprehensive or gagging patient, in a child, and in the temperamental singer this is impracticable if not often impossible.

In order to make cinelaryngoscopy similar to mirror laryngoscopy, a technique has been developed which is based upon the principle of a manipulable rather than a fixed laryngeal mirror. In our technique (Fig. 2) the physician holds the mirror in his hand, introduces it into the pharynx in the usual manner of mirror laryngoscopy, visualizes the larynx, and then photographs at high-speed what he wishes to record. To accomplish this certain changes had to be made in the relationship of the camera and light source to each other and to the patient. The camera was mounted overhead at no sacrifice of accessibility or fixation. The light source was placed beneath the physician's seat and channeled via an enclosure to the larynx. The observer visualizes the larynx at all times through an optical view finder (Fig. 3) while most of the light from the laryngeal mirror is deflected upward into the camera. When the physician sees what he wishes, he steps on a footswitch which triggers the camera and increases the light intensity simultaneously. The laryngeal image remains clearly visible to the physician even during the period of actual shooting. The only limitation to the subject's movements is a flat head rest, thus allowing him to slip aside if he is uncomfortable.

MATERIALS

Camera. The camera incorporated in this unit is the Wollensak "Fastax" WF-4S. It holds either 100 or 400 foot rolls of 16 mm film and is capable of stop-start runs at speeds up to 8000 frames per second.

Light Source. Since only one light source can be used in this particular arrangement, it must be very intense and at the same time relatively cool. Of the many lamps tested the Mole-Richardson Xenon lamp proved the most satisfactory. Although described as a cool light, its proximity to the subject produces too much heat for comfort. In addition, ultraviolet rays are emanated, and so special glass filters have been interposed to absorb both heat and rays. The

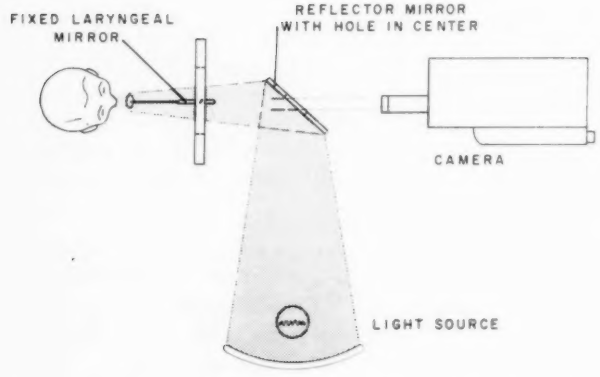

Fig. 1.—Diagrammatic sketch of the technique developed by the Bell Telephone Laboratories for high-speed cinematography of the larynx.

lamp is battery operated and has excellent color balance for daylight color film. When focusing and viewing, it produces an intensity of 17,000 foot candles in the area of the pharynx. A switch (Fig. 4-R) activated by the right foot intensifies the light to 275,000 foot candles at the instant of shooting. This flash is synchronized to trigger the camera. The burst of light can be set for a duration of 1½, 3 or 5 seconds. Eighteen volts D.C. powers the unit for viewing and focusing, and 42 volts D.C. for the bright flash. The control panel (Fig. 4-S) contains the circuit for the lamp plus the synchroflash mechanism.

PATH OF ILLUMINATION AND REFLECTION

Light is directed from its source in the Xenon lamp (Fig. 4-A) to the vocal cords in the following manner: The beam is focused to its narrowest angle and is first directed to a mirror (Fig. 4-B). After experimentation it was decided to utilize a first surface mirror here because light passing through an ordinary mirror to the silver backing and then out again suffers an alteration of color balance and significant decrease in its intensity. This first surface mirror is set at 45° angle and directs the beam of light upward through a tunnel formed by sheet metal (Fig. 4-C). The tunnel insures unobstructed passage of the light beam and shields the eyes of both doctor and subject. Near the top of the tunnel is a heat absorbing glass filter (Fig. 4-D).

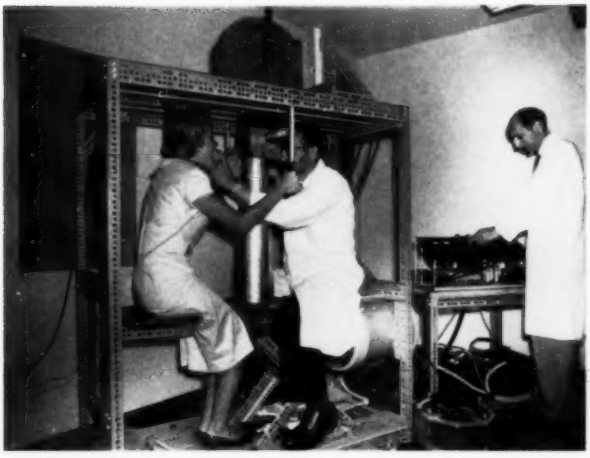

Fig. 2.—High-speed unit showing over-all appearance and relative positions of physician and subject.

A circulating water filter was used originally but, while effective in cooling, it at the same time significantly reduced the light intensity. The beam of light then strikes a specially ground concave first surface mirror (Fig. 4-E) to narrow it further. The mirror is set at a 45° angle and so returns the beam to a horizontal plane. It also has a hole in its center, the purpose of which will be explained shortly. The light beam now enters the mouth, strikes the laryngeal mirror (Fig. 4-F) and is directed downward to illuminate the vocal cords (Fig. 4-G). The diameter of the beam in the pharynx is approximately two inches, and the laryngeal mirror may be held anywhere within this limited but larger than mirror size area during filming. After being illuminated by light deflected from the laryngeal mirror, the image of the vocal cords returns by the same mirror and passes through the concave mirror (Fig. 4-E) which has a hole cut in its center. The purpose of this hole is to permit the reflected image to pass through to a beam splitter (Fig. 4-T). The light in its path from concave mirror (Fig. 4-E) to larynx may be encroached upon by teeth, tongue, etc. The most usable and constant light therefore is that closest to the center of the beam. Since this hole is in the very center of the light beam, it is essential that it be properly designed for minimal loss of light. The laryngeal mirror held at approximately 45° presents itself as an oval shape, and the best configuration for

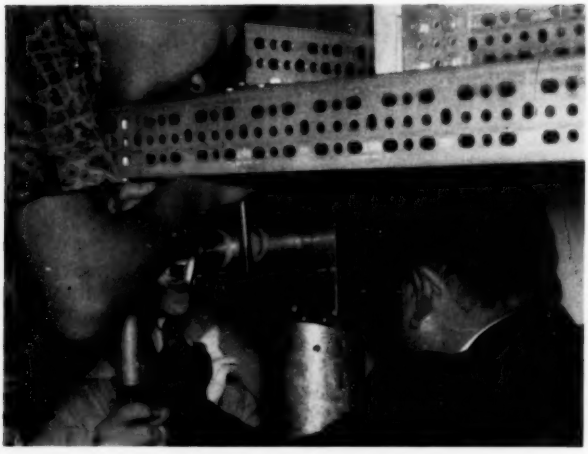

Fig. 3.—High-speed unit in operation showing mirror being introduced and photographer centering image through gun sight.

the hole is also oval. To accomplish this with our set-up a round hole was cut out of the mirror and, when this is tilted to 45°, it takes the shape of an oval. The optimal diameter of the hole in our unit is 25 mm.

When the reflected image passes through the hole and reaches the beam splitter (Fig. 4-T) (a partially aluminized mirror), 99 per cent of the light is reflected upward to the lens of the camera (Fig. 4-H) and 1 per cent continues straight on to an optical view-finder (Fig. 4-I) which gives the physician a magnified view of the larynx. Although the amount of light reaching the eye is only 1 per cent of the total, it affords as much illumination as a head mirror and usual office light source.

The light reflected upward to the camera passes through an optically flat piece of glass (Fig. 4-J) which reflects a fraction of a per cent of light along a tube having a first surface mirror (Fig. 4-K) at its end. A hole in the top of this tube allows the photographer to visualize the laryngeal mirror and image at the same time as the physician.

Since the position of the subject is not stationary, the laryngeal image will not always occupy the center of the light beam. Moreover

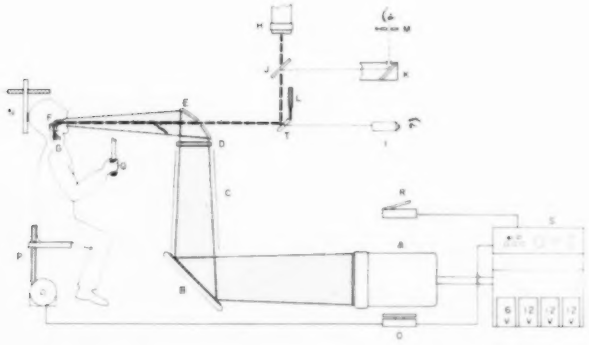

Fig. 4.—Diagrammatic sketch of high-speed photographic unit.

the position of the throat often shifts during actual photography. These factors will be manifested in the film as distracting movements and an off-center larynx. The photographer corrects this by manipulating the handle (Fig. 4-L) of the movable beam splitter (Fig. 4-T) as he observes the larynx through his own mirror arrangement simultaneously with the physician. By means of a gunsight (Fig. 4-M) which permits of finely controlled adjustments, the photographer is able to "zero in" on the light beam as it strikes the beam splitter, detect any deviation in its desired axis, and keep the image centered as it passes upward to the camera.

POSITIONING OF SUBJECT

Given a non-stationary subject, a 135 mm lens, and a shallow depth of field, focusing seemed an enormous problem. The solution, however, proved to be fairly simple. A fixed headrest (Fig. 4-N) provides a definite position for focusing the camera on the vocal cords and yet allows lateral and rotary freedom of movement of the head without altering the focal distance. The headrest unit consists of a board and screw assembly attached to the top of the frame. The back of the patient's head is in contact with the board at all times. This gives the patient a feeling of freedom from enclosures or clamping devices. He may move his head and slip away from the laryngeal mirror should there be any discomfort. While focusing, the patient's height can be rapidly adjusted by a reversing foot switch (Fig. 4-O) controlled by the physician which activates a motorized seat (Fig.

Fig. 5.—Normal vibratory pattern at 6000 frames per second

4-P). Two hand rests (Fig. 4-Q) are provided which the patient grasps for support. Switch (Fig. 4-R) controlled by the physician's right foot, starts the filming sequence by closing a circuit to the control panel (Fig. 4-S).

CONSTRUCTION

The framework for the assembly is constructed of a processed metal known commercially as slotted angle. It superficially resembles

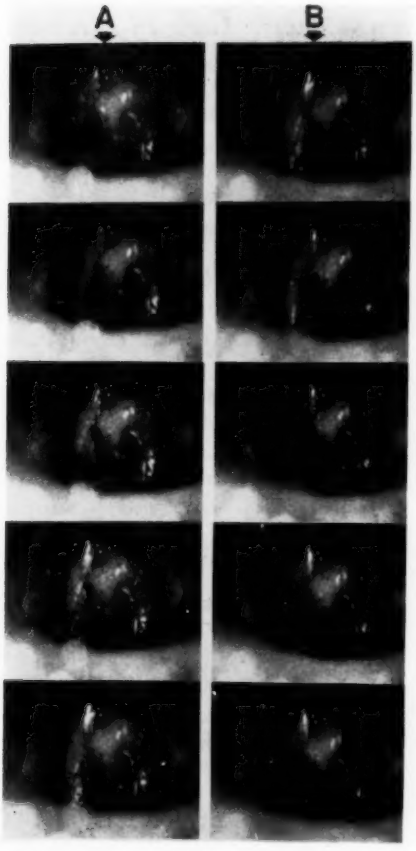

Fig. 6.—Infiltrating carcinoma of vocal cord interfering with normal mobility pattern.

the parts in a child's erector set. The chief advantage of this material is the ease of attaching any number of components to the unit by making use of the recurring hole pattern. Its versatility saved many dollars and untold hours of labor when compared with any other conventional structure. The proper lengths of slotted angle are quickly cut utilizing an angle cutter, and with the insertion of nuts and bolts in the recurring holes a strong bond is accomplished.

FILM

For black and white photography we found Dupont Superior No. 4 to be the most satisfactory. It has an ASA rating of 320. For color we use Kodak Reversal Color Film SO 260, ASA rating 160.

Initially we used black and white film in 100 foot rolls, but at speeds of 4000-5000 frames per second this is exposed in about one second and is therefore suitable for isolated phonatory bursts only. The study of vocal phenomena such as the falsetto "break" and coordinated registration, or the mechanism of laryngeal closure in recurrent nerve palsy, requires a longer shooting time in order to capture the voice in continuity. This is either impossible with a one second exposure or requires such split second timing as to be impracticable because of the gross waste of film. For situations in which it is necessary to photograph a phonatory sequence we utilize film in 400 foot rolls. This allows three seconds of shooting time at 8000 frames per second and more at lesser speeds. Color film is used in 100 foot rolls only because the real value of color lies in demonstrating lesions and, for these, short phonatory bursts are sufficient. Color is not necessary for the study of laryngeal dynamics and also involves far greater expense.

SPEED

For ordinary vocal sequences speeds of 4000-5000 frames per second are adequate. However in studying the upper reaches of the voice, particularly of the female, and in the child, greater speed is necessary to effect slower movement of the vocal cords and permit close study of each vibratory phase. In these situations we shoot at 7000-8000 frames per second. It becomes imperative at these speeds that all available light be utilized to its maximum. Great care must therefore be exercised that such obstructing factors as an incompletely open mouth, prominent tongue or teeth, gauze around the tongue, a partially overhanging epiglottis, etc., do not encroach unduly on the light beam and critically reduces its intensity. These will occur without the operator's being aware of it, and only experience enables him to know what exposure is adequate for best photography at highest speeds.

SOUND RECORDING

A tape recorder is synchronized with the camera, and a small throat microphone is secured comfortably to the subject's neck over

one ala of the thyroid cartilage. This enables the voice to be recorded without background noise.

The principal advantages of this new technique are threefold: 1) It makes available for high-speed photography subjects who otherwise could not be photographed at all or, at best, with difficulty. Simulating the type of laryngeal examination employed by the laryngologist as a routine office procedure overcomes the problems attendant upon bringing the subject over a fixed laryngeal mirror and enables virtually any larynx which can be well visualized by mirror to be photographed at high speed. 2) Because the larynx remains in view during the entire period of actual shooting, it is possible to extend the scope of a study to include vocal sequences as well as single sounds, and film in rolls of 400 feet can be utilized. 3) The increased light intensity resulting from efficient use of a more powerful source permits speeds up to 8000 frames per second and consequent clear delineation of individual vibratory phases in the highest reaches of the voice.

Utilizing this technique the action of the larynx in a wide range of normal and pathological situations is being studied. Figure 5 illustrates fundamental registration in a female vocalist. Figure 6 demonstrates fixation of one vocal cord by infiltrating carcinoma and normal vibratory activity of the other. Recently reported are an evaluation of the neurochronaxic theory[4,5] and the mechanism of the falsetto.[6] Motion pictures on the neurochronaxic theory, the falsetto, and the mechanism of the larynx in health and disease have been produced.

CONCLUSIONS

1. A new technique is described for photographing the larynx at high speed.

2. By this technique the potentialities of high-speed photography in studying the behavior of the larynx in health and disease are greatly enhanced.

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REFERENCES

1. Farnsworth, D. W.: High-Speed Motion Pictures of the Human Vocal Cords. Bell Lab. Records 18:203, 1940.

2. Moore, P., and von Leden, H.: Sound Film, The Larynx and Voice. The Function of the Normal Larynx, 1956.

3. von Leden, H., and Moore, P.: Sound Film, The Larynx and Voice. Laryngeal Physiology under Daily Stress, 1958.

4. Rubin, H. J.: The Neurochronaxic Theory of Voice Production—A Refutation. A.M.A. Archives of Otolaryngology 71:913, 1960.

5. Rubin, H. J.: Further Observations of the Neurochronaxic Theory of Voice Production. A.M.A. Archives of Otolaryngology 72:207, 1960.

6. Rubin, H. J., and Hirt, C. H.: The Falsetto—A High-Speed Cinematographic Study. Laryngoscope 70:1305, 1960.

LXXXIII

BIOCHEMICAL CHANGES IN THE INNER EAR INDUCED BY INSULIN, IN RELATION TO THE COCHLEAR MICROPHONICS

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In recent years, it has been found that administration of insulin produces a waning of cochlear function. Some of these findings cannot be satisfactorily explained unless one assumes that circulatory failure is the main factor in depression of the cochlear microphonics. According to Fernández,[1] and Fernández and Brenman,[2] effect of hypoglycemia induced by insulin on the cochlear microphonics in cats to sound stimuli remained unchanged at blood sugar levels as low as 5 mg per cent. However, the cochlear microphonics diminished in amplitude whenever blood pressure was allowed to fall to low values, and returned to nearly normal when it was restored. They mentioned that this observation suggests that carbohydrates are not essential for cochlear function or that the structure contains a large store of them.

Also, Wing,[3] Konishi and associates,[4] and Muta[5] suggested that the amplitude of the cochlear microphonics from one time to another after administration of insulin does not reflect exhaustion or replenishment of carbohydrates needed for its generation, and that circulatory failure is the main factor in depression of cochlear microphonics.

In the earlier experiments,[6-8] we have observed that depression of the cochlear microphonics in cats was produced after blood sugar had been lowered by insulin, and that this depression generally reversed after intravenous glucose or L-glutamate. However, we were puzzled

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from the beginning of our experiments by the unexpected frequency with which the cochlear microphonics in cats remained unchanged at low levels of blood sugar, and with which depression of the cochlear microphonics in guinea pigs were observed at rather normal levels of blood sugar. These findings also suggest that depression of blood sugar level is not the main factor in depression of cochlear microphonics.

As the result of thermodynamic studies on the energy-producing system in the inner ear,[6-9] it was concluded that both oxygen and glucose are the essential factors in the maintenance of the stationary state of this system. The concentration of glucose of the inner ear fluids, being at least 50 mg per cent, is markedly higher than that of oxygen, even if the inner ear fluids were saturated with oxygen. Comparing with an equivalent revealed several hundred folds in the amount of glucose. Thus, it was assumed that oxygen is the limiting factor in maintenance of the stationary state of energy-producing system.

Therefore, it is reasonable to suppose that depression of the cochlear microphonics induced by insulin is not brought about by glucose deprivation, but by oxygen deprivation in the inner ear. The main purpose of the experiments described here was to evaluate the biochemical changes in the inner ear after administration of insulin, in relation to the cochlear microphonics.

METHODS

Guinea pigs, weighing about 300 g, and rabbits, weighing about 1.5 kg, were used for the present experiments. The animals were deeply anesthetized with an appropriate intraperitoneal dosage of a solution of nembutal.

Measurement of Oxygen Tension in the Inner Ear. The techniques of recording the oxygen tension in the inner ear has been described elsewhere.[10] A platinum micro-electrode was inserted into the scala vestibuli of the basal turn of guinea pig cochlea. In the rabbit, this electrode was inserted into the scala tympani of the basal turn.

Measurement of the Cochlear Microphonics. Incision was made through the skin in the mandibular region of the guinea pig, and the surface of the bulla was exposed, and with a chisel an opening was made to expose the cochlea. The recording electrode was enamel-insulated steel wire of about 200 μ. The tip was introduced into the scala vestibuli of the basal turn of cochlea by means of a micromanipu-

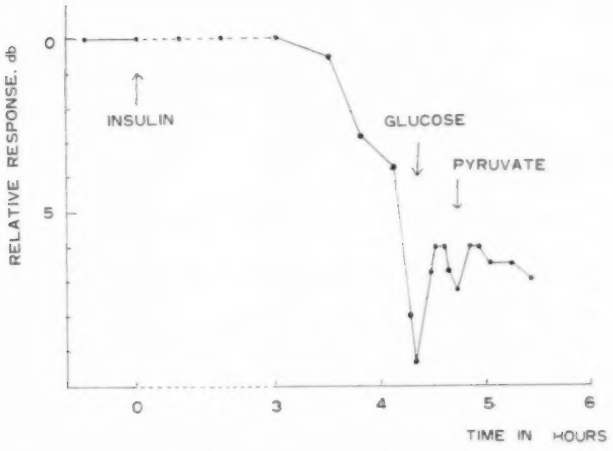

Fig. 1.—Depression of the cochlear microphonics induced by administration of insulin, and their temporary recoveries after administration of substrates. The intraperitoneal dosage of the substrates are as follows: glucose, one ml of 40 per cent solution; sodium pyruvate, one ml of 15 per cent solution; sodium fumarate, one ml of 15 per cent solution. These dosages were given to guinea pigs in all the experiments.

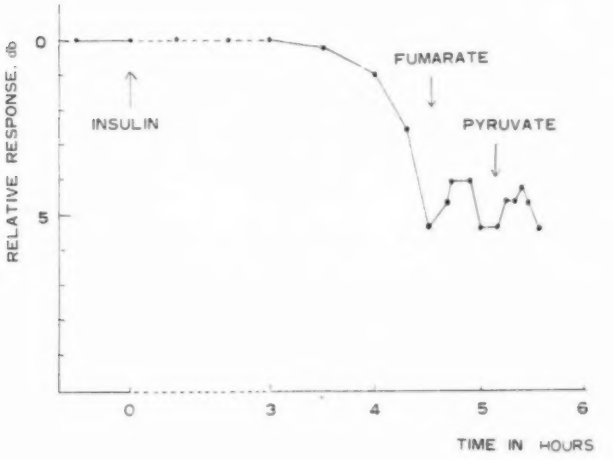

Fig. 2.—Depression of the cochlear microphonics induced by administration of insulin, and their temporary recoveries after administration of fumarate or pyruvate.

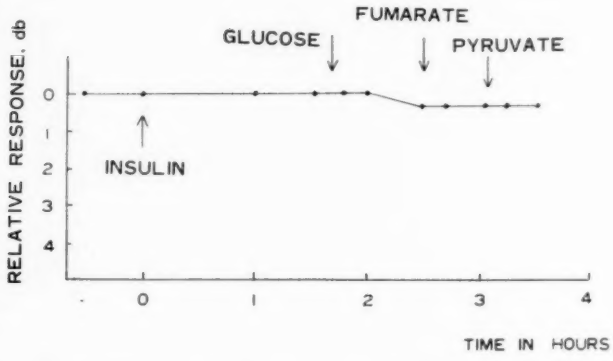

Fig. 3.—Effects of the substrates on the cochlear microphonics in the stationary period after administration of insulin.

lator. The experiments were carried out in a soundproof, electrically-shielded room. The sound (4000 cps) was conducted to the animal's ear by a loud-speaker driver unit mounted outside the shield.

The micromethod described by Klendshoj and Hubbard[11] was used for blood sugar analysis of the insulinized animals.

The open-tube micro-respirometer (Kunningham-Kirk type) was used for measurement of oxygen uptake of the cochlear tissues (the stria vascularis and spiral ligament). These techniques have been described elsewhere.[12] Oxygen uptake represents oxygen disappearance measured in μl/mg dry weight of tissue.

RESULTS

Effect of Administration of Insulin on the Cochlear Microphonics in Guinea Pig. In the first series of experiments, insulin was given in single doses from 8 to 10 unit/kg. In all preparations receiving insulin, the amplitude of the cochlear microphonics persisted at a constant level for as long as six hours. However, in the second series of experiments, it was revealed that a large dose of insulin always causes a progressive deterioration of cochlear function.

Figure 1 shows the result obtained on one guinea pig treated with insulin in a dose of 80 unit/kg. The cochlear microphonics

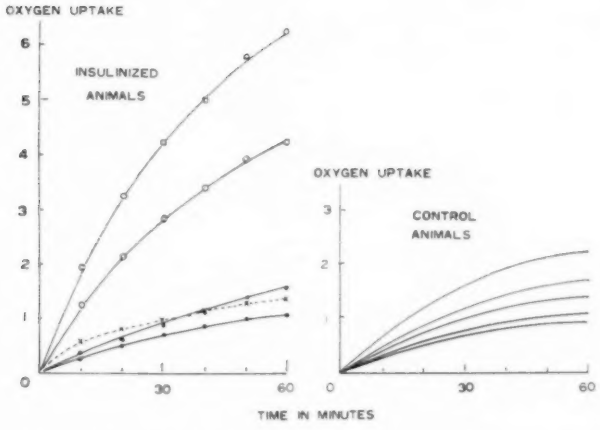

Fig. 4.—Effect of administration of insulin on oxygen uptake of the cochlear tissues. •, two hours after insulin administration; x, three hours after administration; o, six hours after administration.

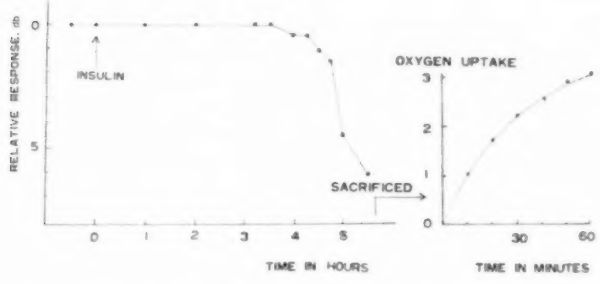

Fig. 5.—Effect of administration of insulin on the cochlear microphonics and oxygen uptake of the cochlear tissues.

began to diminish after three hours of administration of insulin, and for the next 80 minutes the cochlear microphonics showed a loss of 9 db. In the first trial, glucose was administered intraperitoneally, and the cochlear microphonics reversed temporarily. A second trial with pyruvate also caused a temporary increase in the cochlear microphonics.

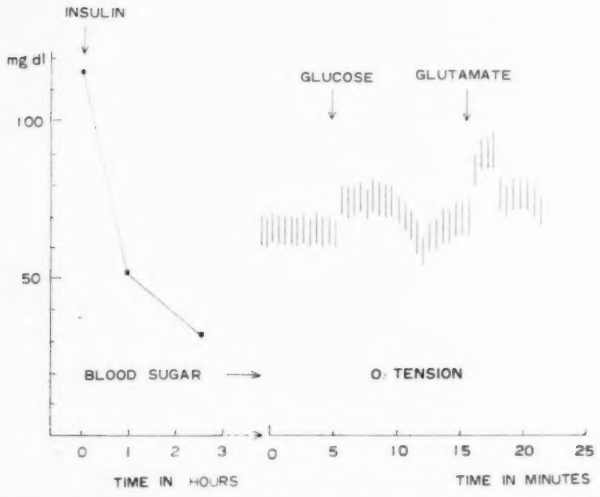

Fig. 6.—Effects of administration of substrates on the oxygen tension in the inner ear of insulinized rabbit. The intravenous dosages of the substrates are as follows: glucose, 3 ml of 40 per cent solution; sodium L-glutamate, 3 ml of 15 per cent solution.

Figure 2 shows another experiment in which insulin, in a dose of 80 unit/kg, was given. In this experiment, the depression of the cochlear microphonics reversed after administration of either fumarate or pyruvate.

Although administration of substrates, such as glucose, pyruvate, and fumarate, recovered the cochlear microphonics in the above experiments, those substrates had no effects, when the amplitude of the cochlear microphonics had not yet diminished (Fig. 3).

These experiments with guinea pigs suggest a possibility: the exhaustion of carbohydrates or Krebs cycle substrates is the main factor in depression of the cochlear microphonics. However, the fact was not simple. The chemical analysis for blood revealed that the concentration of glucose and Krebs cycles substrates do not decrease to the limiting extent in the animals treated with insulin. That is, the blood sugars determined when depression of the cochlear microphonics in guinea pigs occurred, were not allowed to fall below 60 mg %. Furthermore, it was revealed that rabbits treated with insulin

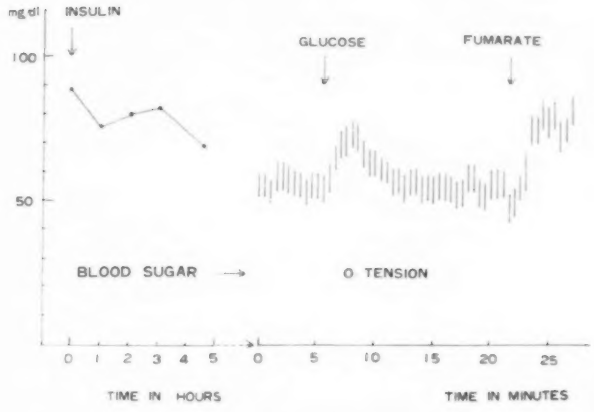

Fig. 7.—Effects of administration of substrates on the oxygen tension in the inner ear of an insulinized guinea pig.

did not show significantly low blood pyruvate levels, while blood α-ketoglutarate levels increased slightly.

This demonstration, that the blood substrate levels in animals did not decrease to the limiting extent, raises the question as to whether depression and recovery of the cochlear microphonics can be totally explained on changes in blood substrate levels. Therefore, this problem must be studied from other angles. It was already described that oxygen is the limiting factor in maintenance of stationary state of the inner ear. Then, it appears that there are the following possibilities: 1) the lack of sufficient oxygen uptake at the cellular level occurs after administration of insulin; 2) the oxygen supply to the inner ear is reduced after administration of insulin.

Changes in the Oxygen Uptake of the Cochlear Tissue After Administration of Insulin. In the first experiments, oxygen uptake of the cochlear tissue of guinea pigs was determined. The animals were killed by decapitation after 2, 3, and 6 hours of administration of insulin, in dose of 80 unit/kg. Then, the cochlear tissues were taken out rapidly at 0° on cracked ice, and placed in the open-tube submicro-respirometer. The medium consisted of Krebs-Ringer phosphate solution containing 10 mM citrate (pH. 7.2). After temperature equilibration at 37° in the thermostat, oxygen uptake was determined in the usual way. The intact animals were used as control.

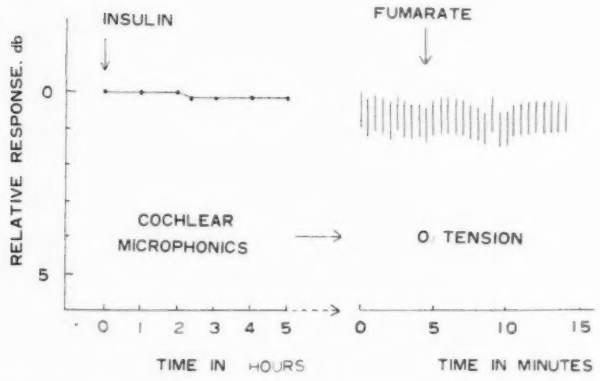

Fig. 8.—Effect of administration of fumarate on the oxygen tension in the inner ear in the stationary state after administration of insulin.

In Figure 4 are given the data from the first series of experiments. There was a good utilization of citrate in the tissues, and inhibitory effect of insulin on the oxygen uptake was not obtained. Especially, with the animals sacrificed after 6 hours, the stimulating effect of insulin was indicated.

In the next experiments, attempt was made to set up conditions which might be more favorable to manifestation of the insulin effect. This consisted in determining oxygen uptake of the tissue when the cochlear microphonics diminished in amplitude.

Figure 5 shows the data in such an experiment. In this experiment, 80 unit/kg of insulin was administered to the guinea pig, and after 4 hours the cochlear microphonics began to diminish. When after 5 hours the cochlear microphonics showed a loss of 6 db, the animal was killed by decapitation. Then the cochlear tissues were taken out rapidly, and oxygen uptake of the tissues was determined. As shown, oxygen uptake of the tissues was not inhibited, but rather stimulated.

There are experiments in the recent literatures showing that insulin is concerned in oxidative reactions, particularly of pyruvate. Krebs and Eggleston[13] stated that insulin acts as a catalyst in the Krebs cycle. According to Stadie and associates,[14] significant increase of pyruvate utilization was observed in the insulin-treated preparation.

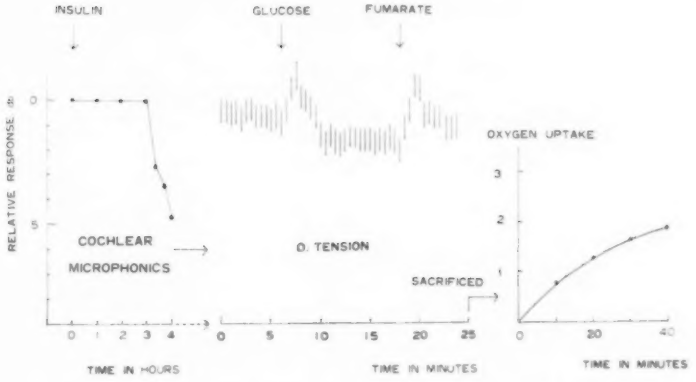

Fig. 9.—Changes in the inner ear after administratoin of insulin.

Further evidence must also be considered. Villee and Hastings[15] reported that the amount of pyruvate metabolized to carbon dioxide was less in muscle, but this was restored to normal by the addition *in vitro* of insulin, and that the total utilization of pyruvate was also decreased in diabetic muscle and brought back to normal when insulin was added. Several general reviews on various aspects of insulin chemistry are in the literature. Among these is the good review by Stadie.[16] According to Stadie, insulin promotes the metabolic transformation of glucose, and oxidative reactions, and increases the efficiency of oxidative phosphorylation.

The stria vascularis and spiral ligament are not difficult to obtain in sufficient quantities to determine their oxygen uptake, but much greater technical difficulties have been encountered in obtaining samples of the hair cells of the organ of Corti. Therefore, the authors were not equipped to determine oxygen uptake of the hair cells throughout the entire series of experiments. However, according to all evidences, it is believed that the metabolic activities in the hair cells also are not inhibited by the administration of insulin.

In conclusion, it must be considered extremely unlikely that lack of sufficient metabolic activities at the cellular level produces depression of the cochlear microphonics in insulinized animals.

Changes in the Oxygen Tension in the Inner Ear after Administration of Insulin. Following administration of insulin, the blood

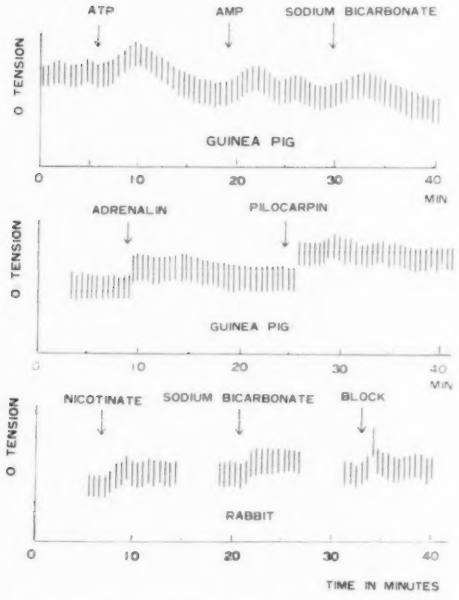

Fig. 10.—The agents, improving the oxygen supply to the inner ear. In the guinea pigs, the following dosages were given intraperitoneally: ATP, 0.02 mg; AMP, 0.02 mg; sodium bicarbonate, 0.5 ml of 7 per cent solution; adrenalin, 0.02 mg; pilocarpin, 0.2 mg. In the rabbits, the following dosages were given intravenously: sodium nicotinate, 10 mg; sodium bicarbonate, 2 ml of 7 per cent solution.

sugar levels in the rabbit began to fall immediately, and around 3 hours the blood sample was 31 mg per cent (Fig. 6). At this time, the cochlea was exposed by opening the bulla, and then the oxygen tension in the inner ear was recorded by means of the oxigraph. Under this condition, the oxygen tension in the inner ear increased markedly after intravenous glucose or L-glutamate.

A similar experiment on a guinea pig treated with insulin in a dose of 80 unit/kg is shown in Figure 7. Although a slow, progressive diminution of the blood sugar was observed with insulin, the blood sugar was not allowed to drop below 68 mg per cent for a longer period than 5 hours. However, there were also the marked effects of administration of either glucose or fumarate on the oxygen tension in the inner ear.

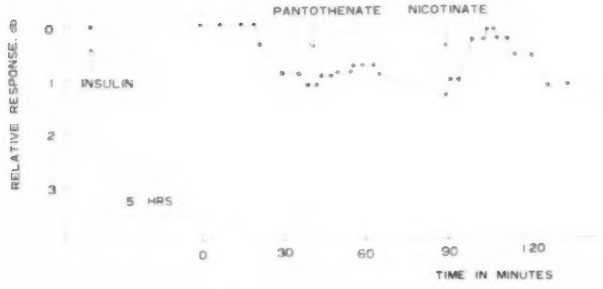

Fig. 11.—Changes in the cochlear microphonics in the insulinized guinea pig after administration of nicotinate and/or pantothenate. The dosages are as follows: sodium nicotinate, 5 mg; pantothenate, 5 mg.

These effects of administration of substrates were not observed for a preliminary period in all the animals treated with insulin, but for a latter period. For example, for a preliminary period from 30 to 120 minutes, glucose had no effects on the oxygen tension.

According to the above data, it is expected that the cochlear microphonics in the animals treated with insulin remain unchanged until the oxygen tension in the inner ear decreases.

Insulin in dose of 80 unit/kg, was administered to the guinea pig. In this animal, the cochlear microphonics remained unchanged for as long as 5 hours. Around 5 hours, the steel wire electrode, used for recording the cochlear microphonics, was removed, and the platinum micro-electrode was advanced into the cochlea in order to record the oxygen tension. In this experiment, administration of fumarate had no effects on the oxygen tension (Fig. 8).

When the cochlear microphonics diminished in amplitude, administration of either glucose or fumarate produced marked increase in the oxygen tension. After recording the data of the oxygen tension, the guinea pig was sacrificed, and the cochlear tissues were taken out rapidly, and the oxygen uptake of the tissues was determined. In this ear with depression of both the cochlear microphonics and the oxygen tension, it was also indicated that the oxygen uptake of the cochlear tissues were emphatically normal (Fig. 9). Therefore, it is considered that oxygen deprivation may be the main cause of deterioration of the cochlear function.

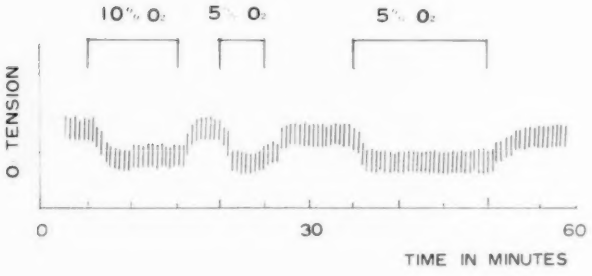

Fig. 12.—Changes in the oxygen tension in the inner ear caused by oxygen deprivation.

Recovery of the Cochlear Microphonics in the Guinea Pigs Treated with Insulin, by Administration of Some Agents. Additional information concerning the effect of oxygen supply on the cochlear microphonics in the animals treated with insulin was obtained by administration of some agents. As agents to increase the oxygen tension in the innear ear, nicotinate, pilocarpin, adenosine triphosphate (ATP), adenylate (AMP), adrenalin, and sodium bicarbonate were injected intraperitoneally or intravenously into the animals (Fig. 10). It was also observed that stellate ganglion block produces an increase in the oxygen tension.

In the following experiment, nicotinate was used. Figure 11 shows the effect of nicotinate on the cochlear microphonics in the guinea pig treated with insulin. Insulin in a dose of 80 unit/kg was administered. After 5 hours, the cochlear microphonics began to fall. Under this condition, administration of nicotinate produced a significant increase in the cochlear microphonics, while a metabolic cofactor, such as pantothenate, had no marked effects. The same results were also obtained by administration of other agents, viz., pilocarpin, and ATP, instead of nicotinate.

Therefore, it may be concluded safely that decrease in effective oxygen supply to the inner ear is the most responsible for depression of the cochlear microphonics induced by administration of insulin. And, in agreement with the results obtained by the previous investigators,[1-5] we consider that circulatory failure is the main factor in decrease in the oxygen supply to the inner ear.

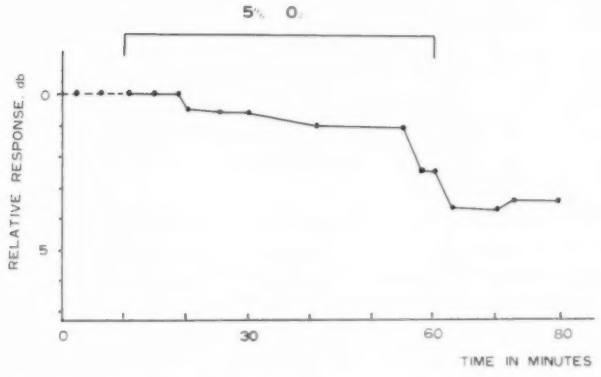

Fig. 13.—Changes in the cochlear microphonics caused by 5 per cent oxygen.

However, decrease in the oxygen tension in the inner ear does not always produce depression of the cochlear microphonics. In the following experiments, two oxygen-deficient mixtures were used. These mixtures contained 10 per cent and 5 per cent oxygen. Oxygen deprivation was introduced by letting the animal breathe the oxygen-deficient mixtures. As shown in Figure 12, the oxygen tension in the inner ear of the guinea pig decreased immediately when either 10 per cent or 5 per cent oxygen was inhaled by the animal. However, the cochlear microphonics did not diminish by inhalation of 10 per cent oxygen, while they diminished only slightly by the inhalation of 5 per cent oxygen after a longer period of time (Fig. 13).

Therefore, it may be assumed that, although oxygen is the limiting factor in maintenance of the stationary state of energy-producing system, far more oxygen than needed is supplied to the inner ear, and that the inner ear can resist to a considerable extent the circulatory failure.

The decrease in the oxygen tension in the inner ear of animals treated with insulin is probably due to the failing blood pressure. However, it may be considered that no significant changes in the cochlear microphonics occur until the oxygen tension in the inner ear falls below critical level. This concept is also supported by the fact[17,18] that the cochlear microphonics are not affected until oxygen deprivation becomes so severe that breathing and circulation begin to fail.

SUMMARY AND CONCLUSION

The cochlear microphonics in the guinea pig were affected by administration of a large dose of insulin, although the blood sugar did not fall below critical level. This depression was consistently recovered when either glucose, pyruvate, or fumarate was administered. The same results also were obtained by administration of the vasodilating agents, viz., nicotinate, ATP, and pilocarpin.

The oxygen tension in the inner ear also decreased after administration of a large dose of insulin, and was consistently reversed by administration of either glucose, L-glutamate, or fumarate.

The manometric studies have revealed that oxygen uptake of the inner ear tissues was not depressed in the animals treated with insulin, even if the cochlear microphonics were affected, and that depression of the cochlear microphonics consistently accompany depression of the oxygen tension in the inner ear.

As a result, it is concluded that the cochlear microphonics do not diminish in amplitude until the oxygen tension falls below the critical level.

NIIGATA UNIVERSITY

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REFERENCES

1. Fernández, C.: The Effect of Oxygen Lack on Cochlear Potentials. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 64:1193, 1955.

2. Fernández, C., and Brenman, A.: Effect of Hypoglycemia on Auditory Cortex and Cochlear Receptor in the Cat. Amer. J. Physiol. 188:249, 1957.

3. Wing, K. G.: Studies of Basic Cochlear Physiology and the Energy-Metabolism of the Cochlear Response in the Cat. Acta Oto-laryng. Suppl. 148, 1959.

4. Konishi, T., Muta, O., and Kanno, T.: The Effect of Hypoglycemia on Cochlear Microphonics. Jap. Jour. Otol. Tokyo 62:Suppl.:220, 1959.

5. Muta, O.: The Effect of Hypoglycemia on Cochlear Microphonics. Jap. Jour. Otol. Tokyo 62:2141, 1959.

6. Koide, Y.: Biochemical Studies on the Labyrinthine Function. Oto-Rhino- and Laryngological Clinic, Kyoto, 50:377, 1957.

7. Koide, Y.: Introductory Studies of the Chemical-Physiology of the Labyrinth. Acta Med. et Biol. 6:1, 1958.

8. Tajima, S., and Koide, Y.: Effects of Insulin Injection upon the Cochlear Microphonics. Oto-Rhino- and Laryngological Clinic, Kyoto, 52:1384, 1959.

9. Misrahy, G. A., Hildreth, K. M., Shinabarger, E. W., Clark, L. C., and Rice, E. A.: Endolymphatic Oxygen Tension in the Cochlea of the Guinea Pig. J. Acoust. Soc. Amer. 30:247, 1958.

10. Koide, Y., Yoshida, M., Konno, M., Nakano, Y., Yoshikawa, Y., Nagaba, M., and Morimoto, M.: Some Aspects of the Biochemistry of Acoustic Trauma. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1960, in press.

11. Klendshoj, N. C., and Hubbard, R. S.: A Method for the Determination of Sugar in Small Amounts (0.02 cc) of Blood. J. Lab. and Clin. Med. 25:1102, 1940.

12. Yoshida, M., and Koide, Y.: Effects of Anaerobic Incubation on the Respiration of the Inner Ear Tissues. Oto-Rhino- and Laryngological Clinic, Kyoto, 53:33, 1960.

13. Krebs, H. A., and Eggleston, L. V.: The Effect of Insulin on Oxidations in Isolated Muscle Tissue. Biochem. J. 32:913, 1932.

14. Stadie, W. C., Haugaard, N., and Perlmutter, M.: The Effect of Insulin upon Pyruvate Utilization by Pigeon Muscle. J. Biol. Chem. 172:567, 1948.

15. Villee, C. A., and Hastings, A. B.: The Utilization *in vitro* of C^{14}-Labelled Acetate and Pyruvate by Diaphragm Muscle of Rat. J. Biol. Chem. 181:131, 1949.

16. Stadie, W. C.: Current Concept of the Action of Insulin. Physiol. Rev. 34: 52, 1954.

17. Wever, E. G., Lawrence, M., Hemphill, R. W., and Straut, C. B.: Effects of Oxygen Deprivation upon the Cochlear Potentials. Amer. J. Physiol. 159:199, 1949.

18. Gisselson, L.: The Effect of Oxygen Lack and Decreased Blood Pressures on the Microphonic Response of the Cochlea. Acta Oto-laryng. 44:101, 1954.

LXXXIV

VOCAL REHABILITATION IN A BOY WITH ESOPHAGEAL VOICE

CLINICAL NOTE

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A boy of 12 years was referred for vocal rehabilitation with the following history:

At the age of four years the child underwent a diagnostic direct laryngoscopy in the course of which a biopsy specimen was taken from the posterior wall of the larynx below the level of the vocal cords. During this procedure, through a tragic mishap, a fistula was created between the subglottic part of the larynx and the esophagus. Two attempts at repair of the fistula were unsuccessful and a large tracheo-esophageal perforation persisted. Subsequent perichondritis led to a destruction of the posterior part of the cricoid and of the left arytenoid. In addition, severe stenosis of the larynx and of the upper trachea developed. To protect the airways tracheotomy was done, and feeding was secured through a gastrotomy.

At this point the child was admitted to Mount Sinai Hospital. First, the perforation in the esophagus was closed by surgery (Dr. Garlock). Following this operation, the damage to the larynx and the upper trachea was repaired (Dr. Som). In a series of plastic operations, using grafts, plastic molds and a semilunar section of cartilage from the nasal septum, the lumen was restored and the laryngeal structures were rebuilt. The upper tracheal lumen was restored by excision of the scars and relining of the denuded area with free skin grafts.

At the time of the referral, eight years after the incident, the boy presented a somewhat underdeveloped but otherwise well-formed

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larynx with a left cord that is immobilized in the paramedian position, and a mobile right cord. He still wore the tracheotomy canula since the newly created laryngeal lumen, while sufficient under normal circumstances, might become dangerously narrow in acute laryngitis or in an allergic congestion. It was planned to remove the cannula after laryngeal growth during puberty had assured a safe airway.

From a phoniatric point of view the boy presented a rare phenomenon: without any instruction and without ever having heard alaryngeal voice the boy had developed esophageal speech spontaneously, soon after the incident, and had used it exclusively ever since. He spoke in a squeaky, high-pitched vocie, quite fluently and fairly intelligibly. For at least seven years he had not used his vocal cords for voice, coughing and other forms of glottal closure.

The boy was referred by the surgeon to the present writer for an attempt to revive laryngeal phonation. Aside from the preference of a natural laryngeal voice over esophageal speech it was felt that the restoration of vocal cord function by providing the growth-stimulus that active muscular function exerts on laryngeal growth during puberty was of great importance.

On the other hand, vocal rehabilitation was severely handicapped by the necessity of continuing the wearing of the tracheotomy tube. With the tube in place the air-stream was deflected away from the vocal cords. Removal of the tube was permitted only for brief periods of time in the office; this condition made voice practice at home impossible.

During the practice sessions in the office, a quick and complete closure of the stoma was necessary. The strong subglottic air-pressure required a very firm and air-tight seal. This was achieved by covering the stoma, after removal of the cannula, with a broad adhesive tape and securing it by a few turns of a wide gauze bandage.

The therapeutic problem was very similar to the revival of phonation in a patient with one paralyzed vocal cord, except that the long period of phonatory inactivity during the formative years of the boy had abolished practically all the reflexes that control normal glottal closure.

A number of conventional methods used for the revival of glottal closure and of phonatory activity, such as Froeschel's pushing exercises and the use of the coughing reflex, were tried without any success.

For almost six weeks all attempts to produce phonation resulted only in a noisy blast of air.

Finally, the stimulating effect of vibratory-acoustic sensations was tried. The earpieces of a stethoscope were placed into the ears of the boy, and the diaphragm was held, alternately, against the larynx of the phonating therapist and the larynx of the boy, who was encouraged to imitate the sensation. After a number of unsuccessful attempts suddenly a loud sound was produced, the first laryngeal sound after many years of phonatory inactivity. Persistent training with this device enabled the boy soon to produce voice at will and, finally, unaided by the instrument.

From then on, voice practice became very important. To make this possible the boy was fitted with a special cannula with an oval window in the upper curvature of the tube. During the practice periods, at the office and at home, the outer opening was plugged with a stopper, with the result that the air-stream was then directed against the vocal cords.

At this stage the psychological problem arose of persuading the boy to make use of his "new" voice in ordinary life. Up until this time, upon the advice of the psychiatrist, it had been felt best to minimize the abnormality of the esophageal voice, but now, after consultation with the psychiatrist, it was decided to stress the ugly sound and the stigmatizing character of the esophageal voice and to emphasize the importance of acquiring a voice like that of every normal person.

Further treatment dealt with improvement of breath control, of flexibility of pitch and of melody of speech. During this period puberty set in. Thanks to the revival of vocal cord function the boy went into the period of the change of voice with active laryngeal muscles which provided a stimulus for laryngeal growth. Slowly, the voice dropped by more than an octave.

At the end of this period the cannula was removed and the opening in the neck was covered through a plastic procedure. The patient, now a young man, speaks at present with a still somewhat rough but otherwise satisfactory voice of deep masculine timbre.

SUMMARY

The case of a boy is reported who, at the age of four years, sustained a traumatic laryngo-esophageal fistula with subsequent partial

destruction of the larynx and severe laryngeal stenosis. After plastic reconstruction of the larynx the boy had used esophageal voice for seven years. Revival of vocal cord phonation was achieved by the use of acoustic-vibratory sensations. The case is reported as an example of vocal rehabilitation through the teamwork of the laryngeal surgeon and the phoniatrist.

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LXXXV

TOXICITY OF BRONCHOGRAPHIC CONTRAST MEDIA

AN EXPERIMENTAL INVESTIGATION

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The value of bronchography as a diagnostic procedure is the result of painstaking technical refinement together with cumulative realization of the advantages, indications and limitations of the method. The commendable progress of the past four decades is the more remarkable in that it has occurred despite lack of commensurate improvement in the *sine qua non* of bronchography—the opaque contrast medium. Iodized poppy seed oil, introduced in 1922, has only recently been displaced as the most widely used medium.[1] The number of opaque media developed in recent years is a further indication that, although actively sought, the ideal bronchographic contrast medium has not been attained.

The characteristics of the ideal bronchographic contrast medium are stringent. It must have high radiopacity and the ability to coat bronchial walls uniformly and rapidly. It must not penetrate the alveolar ducts and alveoli (few would still contend that it is essential

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to demonstrate alveolar patency). It must undergo prompt, complete expulsion from the lungs after bronchography. It must be physiologically and pharmacologically inert. No existing medium meets these requirements.

Recent attempts at improvement have followed three differing approaches. First, a non-iodized (hence non-radiopaque) oil has been used as a vehicle for absorbable compounds containing organically-bound iodine. In Dionosil Oily, for example, iodine is bound to an organic ester which is hydrolyzed in the lung and then absorbed as an organic acid without release of iodide in the body. Persistence of the non-opaque oil in the lungs does not by itself interfere with subsequent chest radiography.

A second approach has been replacement of the iodine atom by others of similar atomic weight such as barium. This substitution avoids whatever hazard is created by the administration of iodinated media and eliminates the prolonged contamination of serum iodine levels that occurs after use of conventional bronchographic media. Other advantages claimed for barium are lack of absorption, and low cost.

A third approach has consisted of addition of various thickening agents, such as mucin and carboxymethylcellulose, to bronchographic media. The "high viscosity" media usually are cleared rapidly from the lungs, and seldom enter the alveoli. The conflicting evidence of the toxicity of the carboxymethylcellulose colloid has been reviewed by Dunbar *et al.*[2] A thickening agent widely used in bronchography is powdered sulfanilamide. This material cannot be considered innocuous. It has been shown that administration of a Lipiodol-sulfanilamide suspension such as Visciodol invariably is followed by absorption of sulfanilamide. The resultant blood sulfanilamide concentrations approach, and may exceed, therapeutic blood levels of this drug. In addition a secondary methemoglobinemia usually occurs, at times causing clinical cyanosis.[3]

The present investigation was undertaken to survey the relative toxicity of eight bronchographic contrast media. These include: a) the commercial media Iodochlorol, Dionosil Oily, Lipiodol, and Visciodol, of which the last is a "high viscosity" medium; b) experimental modifications of Iodochlorol and Lipiodol incorporating tristearin or succinylsulfathiazole as thickening agents; and c) a barium sulfate-carboxymethylcellulose suspension, used clinically by Nelson *et al*[4] and similar to that described by Di Rienzo and Periera-Duarte.[5]

TABLE I

BRONCHOGRAPHIC CONTRAST MEDIA EMPLOYED IN INVESTIGATION

ANIMAL GROUP	CONTRAST MEDIUM	DESCRIPTION	RELATIVE VISCOSITY (37° C.)
A	Chloriodized peanut oil (Iodochlorol®) and tristearin	An 0.13% suspension of tristearin (Fisher, Highest Purity) in chloriodized oil	2.35
B	Chloriodized peanut oil (Iodochlorol®) and succinylsulfathiazole	An 0.20% suspension of succinylsulfathiazole powder (Sulfasuxidine, Merck Sharp & Dohme) in chloriodized oil	2.25
C	Chloriodized peanut oil (Iodochlorol®)	Unmodified commercial preparation	1.32
D	Iodized poppyseed oil (Lipiodol®)	Unmodified commercial preparation	1.0
E	Iodized poppyseed oil and sulfanilamide (Visciodol®)	Unmodified commercial preparation (320 mg sulfanilamide per milliliter)	2.45
F	Peanut oil (non-iodized) and propyliodone (Dionosil Oily®)	Unmodified commercial preparation	0.38*
G	Iodized poppyseed oil (Lipiodol®) and tristearin	A 1.3% suspension of tristearin (Fisher, Highest Purity) in iodized oil	2.20
H	Barium sulfate in carboxymethylcellulose	A suspension of 20 gm of barium sulfate (Micropaque®) in 40 ml of a 1.5% solution of Hercules Premium 70 High Sodium carboxymethylcellulose in isotonic saline	Not tested

* At 26° C.

MATERIALS AND METHODS

1. *Animals.* Male and female adult New Zealand white and gray rabbits weighing 1.8 to 2.5 kg were divided into eight groups of 12 each. During the period of study the animals were maintained in animal quarters without special care.

2. *Bronchographic Media.* The eight media employed in this investigation and their relative viscosities are described in Table I. All were administered at room temperature. The barium sulfate-carboxy-

methylcellulose suspension was prepared according to the specifications of Nelson.[6]

3. *Technique of Injection.* Following induction of anesthesia by open drop ether each animal was earmarked and its anterior cervical region was shaved. With the animal in supine position and neck hyperextended, an 18-gauge needle was introduced percutaneously into the trachea 1-2 cm below the larynx. A syringe containing the bronchographic medium was attached to the tracheal needle by a polyethylene catheter also filled with the medium. One ml of the medium was rapidly injected, the syringe and catheter removed, and a second syringe, containing air, attached to the needle. The air was injected, expelling into the trachea any medium remaining in the needle. The animal was then held erect for several minutes; no further anesthesia was given. After chest roentgenography the animal was returned to its cage.

4. *Radiography.* Chest roentgenograms were taken within 2 to 5 minutes after injection of the contrast material. The x-ray film was promptly developed and inspected while wet. If contrast material was demonstrated within the major bronchi on either side the injection was considered satisfactory. Occasionally contrast material was demonstrated in the soft tissues of the neck, the result of undetected penetration of the needle beyond the trachea. In this event the injected animal was replaced by an intact substitute, or utilized for a second injection of the same contrast medium if the animal's condition was judged satisfactory.

Prior to histological study of the lungs (see below), both lungs of each animal were cut in sagittal sections; roentgenograms of these sections were made to demonstrate macroscopic amounts of residual contrast medium.

5. *Histologic Technique.* The animals were killed with intravenous sodium pentothal at 2, 4, 8, 16, 32 and 150 days after injection. A few animals died during the course of the study. The heart and lungs were removed en bloc. A cannula was inserted into the right ventricle or the pulmonary artery. Buffered (acetate) formalin was perfused through the lungs until the flow from the pulmonary veins was clear. The veins were then tied and the lungs expanded to normal size. The cannula was withdrawn as the pulmonary artery was ligated. The lungs and heart were immersed in similar formalin for at least one week. The lungs were then separated and a number of parallel cuts made through them at intervals of about 4 mm.

Blocks of lung were then selected for tissue processing so that usually both lobes of the left lung and usually two of the three lobes of the right lung were included. The tissue was selected so as to include any discolored or firm areas. Usually two blocks of tissue were sufficient. Occasionally, however, up to five blocks were selected. Hematoxylin and eosin stains were made from each block. From selected blocks, Masson's trichrome, Wilder's reticulin, periodic acid Schiff, Brown-Brenn, and toluidine blue stains were made. Sudan IV and Flaming Red stains for fat were done on selected lungs from each series, usually from six animals killed at varying intervals from four days to five months.

6. *Analysis of Roentgenographic and Histological Data.* The chest x-ray films were made to confirm intrabronchial penetration of the contrast medium. The films of the sectioned lungs were analyzed with respect to their content of residual contrast medium. This was graded on a scale of 0 to 4, 0 denoting no visible residuum and 4 denoting a marked residuum.

The histologic sections were examined without knowledge of the media used. They were graded on a scale of 1 to 4 in increasing severity with regard to:

1) Septal exudate (granulocytic or mononuclear or both)

2) Alveolar exudate (granulocytic or mononuclear or both)

3) Abscess formation

4) Scarring

5) Foreign material, lipid or otherwise

The intensity of a reaction and its size and frequency were considered in grading (Fig. 1).

RESULTS

Group A (Iodochlorol plus Tristearin). This combination resulted in a moderately severe mononuclear septal and alveolar exudate and a slight granulocytic exudate which persisted in varying degrees for at least one month. Even at five months foci of macrophages were present in alveoli in 2 of 7 animals. Extracellular lipid was considerable even at 32 days, whereas intracellular lipid was always slight. One small abscess was present in one animal, sacrificed at 16 days.

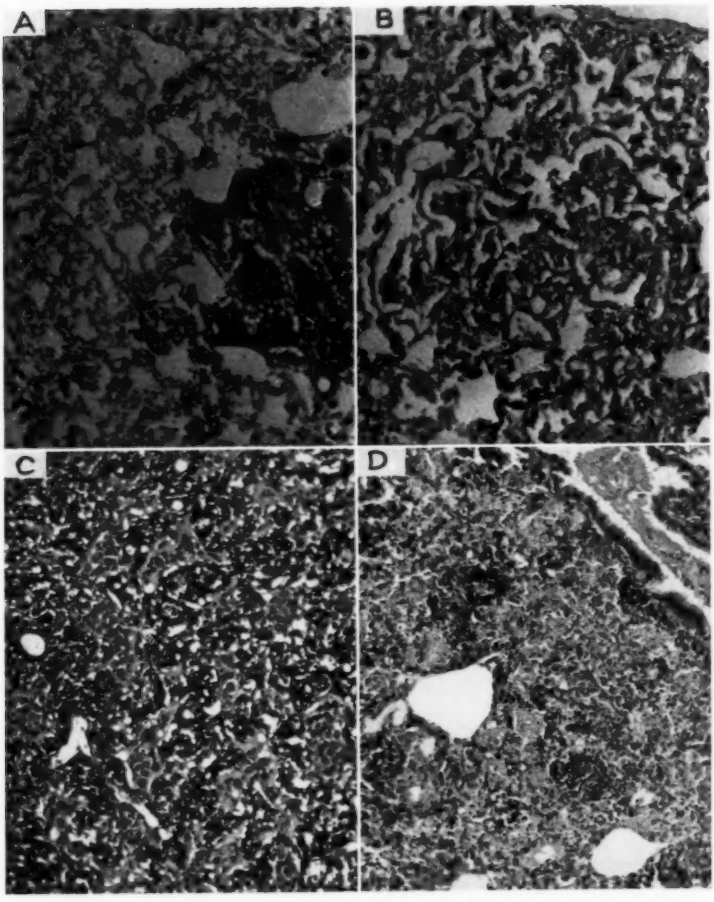

Fig. 1.—A comparison of graded reactions of mononuclear cell exudates in alveolar spaces. *a.* Lungs with occasional foci of intra-alveolar mononuclear cells were graded as 1+ reaction. *b.* Lungs with a more diffuse alveolar exudate of mononuclear cells as illustrated here were graded as 2+ reaction. *c.* Lungs with an alveolar exudate of mononuclear cells such as this were graded as 3+ reaction. *d.* Lungs with an intense alveolar exudate of mononuclear cells as illustrated here were graded as 4+ reaction. Mononuclear cells in the small bronchus as well as in the alveoli contain particulate foreign material. This animal had received barium sulfate-carboxymethylcellulose.

The 7 animals sacrificed at five months had no scars and only 2 had slight alveolar exudate associated with slight amounts of lipid material.

Group B (Iodochlorol plus Succinylsulfathiazole). This combination provoked an initial moderately severe mononuclear exudate principally in the alveolar septa and a less pronounced mononuclear and granulocytic reaction in the alveoli. This exudate decreased in amount in the animals sacrificed in the succeeding 30 days so that at 32 days only a slight mononuclear reaction was present in the septa and the alveoli. Three of 7 animals sacrificed at five months had persistent foci of mononuclear cells, principally in alveoli. Scarring did not occur.

Lipid residue progressively decreased and at five months only small amounts were still present. Again the lipid material, where present, was largely extracellular.

Group C (Iodochlorol). The inflammatory reaction to the materia was moderate in intensity and distribution initially and continued so throughout the first month. One animal developed a severe acute inflammatory reaction with multiple small abscesses. One of 5 animals sacrificed at five months had a large area of mononuclear cell reaction and several smaller foci of similar cells. The other 4 animals had no residual inflammation or scarring.

Extracellular lipid was present in moderately large quantities even at 16 days. Intracellular lipid was always scant.

Group D (Lipiodol). This material provoked a slight inflammatory reaction, acute and granulomatous, in both alveoli and interstitial tissues which disappeared by the 16th day. No scar tissue or residual inflammatory reaction was present in any of the material from the 6 animals examined five months after the instillation.

Lipid material was almost all extracellular and persisted until the 16th day. During this time surprisingly large amounts of lipid materia were present with slight or no inflammatory reaction.

Group E (Visciodol). This material provoked a slight to moderate reaction in both alveoli and alveolar septa, in which both granulocytic and mononuclear cells participated. The material was cleared by the animals without any residual scarring at five months.

The lipid material, noted in the first two weeks, remained extracellular, filling and distending the alveoli.

Group F (Dionosil Oily). Although this material caused only slight amounts of acute and granulomatous inflammatory reaction, some of the material and a relatively large percentage of the inflammatory reactions persisted in the lungs throughout the entire period of five months. At five months the material had assumed an eosinophilic fibrillar structure and stained positively with both Sudan IV and Flaming Red. Multinucleated foreign-body-type giant cells were frequently associated with these structures. No significant scarring occurred despite the long period of inflammatory reaction.

Group G (Lipiodol plus Tristearin). This combination caused a much more severe inflammatory reaction than did Lipiodol alone. The initial moderately severe acute inflammatory reaction had largely disappeared by eight days. A granulomatous reaction, at first of mononuclear cells (at four days) and later (eight days) including multinucleated foreign body giant cells, persisted throughout the period of the experiment. Large abscesses were present in 3 of 7 animals examined at five months. Lipid material was present in and around these abscesses.

Lipid material was abundant at four days and decreased in amount more slowly than did some of the other agents. It too remained largely extracellular. Even when surrounded by clumps of macrophages and multinucleated giant cells, the lipid material was largely extracellular.

Group H (Barium Sulfate plus Carboxymethylcellulose). This combination provoked the most intense initial inflammatory reaction, the largest number of abscesses, the largest amount of scarring, and remained in large quantities in the lungs for the entire term of the experiment. The initial and persistent inflammatory reaction was predominantly an intra-alveolar exudate of mononuclear cells. The macrophages had abundant pale yellowish particulate refractile material in their cytoplasm. Granulocytic cells were prominent throughout the experiment and, of course, were especially prominent in the abscesses.

Carboxymethylcellulose was identifiable in the tissues as a pale acidophilic material with hematoxylin and eosin stains, as pale blue with toluidine blue, as pale yellow with Brown-Brenn, and failed to stain with the periodic acid Schiff and the lipid stains. The material was present in the alveolar spaces of animals until the 14th day after injection. By this time the intensity of staining with toluidine blue had decreased considerably. In tissues examined at the 16th day and

TABLE II

INCIDENCE OF PULMONARY PATHOLOGICAL FINDINGS AT FIVE MONTHS*

EXUDATE		ABSCESS		SCARRING		RESIDUAL FOREIGN MATERIAL	
Group D	0/6	Group D	0/6	Group D	0/6	Group D	0/6
Group E	1/7	Group E	0/7	Group E	0/7	Group E	0/7
Group A	1/7	Group A	0/7	Group C	0/5	Group C	0/5
Group C	1/5	Group C	0/5	Group B	0/7	Group B	2/7
Group B	3/7	Group B	0/7	Group F	0/6	Group A	2/7
Group F	5/6	Group F	0/6	Group A	1/7	Group G	3/6
Group G	3/6	Group H	1/5	Group G	2/6	Group F	5/6
Group H	5/5	Group G	2/6	Group H	3/5	Group H	5/5

* Number of animals with positive findings / Number of animals examined

thereafter, carboxymethylcellulose could no longer be differentiated from protein alveolar exudate. After the second week many macrophages had abundant pale cytoplasm which was pale blue with toluidine blue and largely failed to stain with the periodic acid Schiff technique. This material may have represented intracellular carboxymethylcellulose. However, no cytoplasmic granules, such as described by Hellstrom and Holmgren,[7] were seen.

COMMENT

Table II summarizes the histopathologic findings and grades the media according to increasing toxicity at five months. Since this is a quantitative study, these results cannot be compared directly with those of other investigators, but in the following paragraphs an effort will be made to cite the comparable results of others. An exhaustive review of the literature can be found in the recent paper of Dunbar *et al*,[2] and will not be repeated here.

Lipiodol was the least toxic compound investigated judging by the comparison of the histologic changes at five months. There was no evidence of alteration of the lung structure and no residual oil

was found. Virtually as innocuous as Lipiodol were Visciodol, Iodochlorol-Tristearin, and Iodochlorol alone. None of these media remained in the lung at five months and they differed from Lipiodol only in that a small amount of exudate was present in one animal in each group. However, the viscosity of Iodochlorol was found to vary from lot to lot. Thus a given increment of viscosity could not necessarily be gotten from a constant tristearin or sulfasuxidine concentration.

Iodochlorol-sulfasuxidine and Dionosil Oily were intermediate in toxicity. An oily residue from both compounds was present. Our findings agree with those of Bjork and Lodin,[8] who found oily residue in eight of ten rabbits at three months, but differ from those of Nelson *et al*,[4] who could find no permanent histopathological effects from Dionosil Oily in rabbits at ten weeks. Dunbar *et al*[2] could discover no residual Dionosil Oily at one or at four months in rats, but one animal of the 36 who received Lipiodol had residual oil present. These compounds, particularly the widely used Dionosil Oily, definitely produced more abnormal changes in the lung than Lipiodol.

Lipiodol-tristearin produced a distinctly higher incidence of abnormal findings in the lungs than the compounds mentioned above. This medium was associated with the largest number of abscesses and there was a large residue in five of the six animals studied at five months.

The barium sulfate-carboxymethylcellulose suspension was by far the most toxic. There was more exudate, more scarring, and a larger residue than with any of the other media. One animal had a large abscess.

These results differ from those of Nelson *et al*,[4] who found no evidence of permanent damage in rabbit and human lung tissue after administration of an identical suspension. Others also have noted lack of uniformity in experimental results using carboxymethylcellulose.[9,10] However, our results agree with those of Dunbar *et al*,[2] who demonstrated that barium sulfate in tap water and in dilute sucrose solution produced acute reactions in the rat lung and was retained for longer periods than iodized poppy seed oil.

There are no stains to demonstrate with certainty residual carboxymethylcellulose. The areas of histopathologic change always contained barium sulfate. The present study has not separated changes which may have been due to barium sulfate alone, carboxymethylcellulose alone, or the two in combination.

SUMMARY

A quantitative study of the pathological changes produced in the rabbit lung by various bronchographic media was made. Lipiodol, Iodochlorol, Visciodol, and Iodochlorol-tristearin suspension were the least toxic. Dionosil Oily and Iodochlorol-Sulfasuxidine suspension were slightly more toxic. A Lipiodol-tristearin suspension was moderately toxic. The most toxic medium was a suspension of barium sulfate in carboxymethylcellulose. It was also found that toxicity was not related to the viscosity of the medium and that tristearin was not a satisfactory thickening agent.

Although these results may not necessarily be applicable to humans, they indicate that suspensions of barium sulfate in carboxymethylcellulose are of sufficient toxicity to animals to warrant further investigation before being recommended for human use.

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REFERENCES

1. Report of Committee on Broncho-Esophagology. Bronchography: Summary of a World-Wide Survey. Dis. of Chest 33:251-260 (Mar.) 1958.

2. Dunbar, J. S., Skinner, G. B., Wortzman, G., and Stuart, J. R.: An Investigation of Effects of Opaque Media on the Lungs with Comparison of Barium Sulfate, Lipiodol, and Dionosil. Am. J. Roentgenol. 82:902-926 (Nov.) 1959.

3. Johnson, P. M., and Irwin, G. L.: An Evaluation of the Pharmacological Hazards Resulting from Use of "Visciodol" in Bronchography. Radiol. 72:816-828 (June) 1959.

4. Nelson, S. W., Christoforidis, A., and Pratt, P. C.: Barium Sulfate and Bismuth Subcarbonate Suspensions as Bronchographic Contrast Media. Radiol. 72:829-838 (June) 1959.

5. di Rienzo, S., and Pereira Duarte, R. O.: Die Bronchographie mit Barium. Fortschr. Roentgenstr. Nuklearmed. 86:315-318 (Mar.) 1957.

6. Nelson, S. W.: Personal Communication.

7. Hellström, B., and Holmgren, Hj.: The Reaction of the Lung on Bronchography with Viscous Umbradil (Umbradil-Viskös B) (Astra), Umbradil (Astra), and Carboxymethyl-Cellulose. Acta Radiol. 32:471-485 (Dec.) 1949.

8. Bjork, L., and Lodin, H.: Pulmonary Changes Following Bronchography with Dionosil Oily (Animal Experiments). Acta Radiol. 47:177-180 (Mar.) 1957.

9. Werthemann, A., and Vischer, W.: Zur Frage der Lungenveränderungen nach Bronchographien mit carboxymethylcellulose-haltigen Kontrastmitteln. Schweiz. Med. Wschr. 81:1077-1080 (Nov. 3) 1951.

10. Zollinger, H. U.: Schädigt die Joduron-Bronchographie das Lungenparenchym? Beitrag zur Pathogenese der Schleimgranulome, der xanthomatösen und der interstitiellen Pneumonie bei primären Lungenprozessen. Schweiz. Med. Wschr. 81: 210-216 (Mar. 3) 1951.

LXXXVI

IS CONGENITALLY SHORT ESOPHAGUS TRULY A RARE CLINICAL ENTITY?

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There are certain esophageal abnormalities which, even though detected by x-ray studies, require further investigation for accurate diagnosis. This group includes hiatus hernia, the congenitally short esophagus and peptic esophagitis. After discussion with many radiologists, gastro-enterologists, internists and esophagologists, I was amazed at the different concepts encountered.

The incidence of hiatus hernia has been reported many times with very little agreement. Kirklin and Hodgson[1] reported an incidence of 1% to 2%. Nuzum[2] found 12.2% of 1957 patients had some form of true hiatus hernia. Olsen[3] observed 220 cases over an eleven year period at the Mayo Clinic, or 10% of the cases studied there. In a recent pilot study of 100 consecutive cases Stein and Finkelstein[4] at the Graduate Hospital of the University of Pennsylvania have shown that, considering all types of variations of the degree of displacement of the cardia of the stomach above the diaphragm, the incidence of hiatus hernia may be as high as 50%. They say: "We realize that the criteria for diagnosing small hiatal hernias are at present debatable and subject to various interpretations because of the limited knowledge of the anatomy and physiology of the cardio-esophageal area and the hiatus. Inevitably many of our current concepts will be modified as our understanding of these structures and their functions improves. Nevertheless, anatomic and physiologic disturbances in this region are, in our opinion, quite common, warranting provisional criteria for evaluation which we now offer. There is no difficulty in accurately diagnosing moderate-sized or even small hernias of the para-esophageal type, nor is there usually any problem recognizing those hernias associated with a shortened esophagus. However, the recognition of small hernias of the sliding type frequently presents a challenge because of the difficulty in identifying the diaphragmatic esophageal hiatus and the difficulty in distinguishing a small outpouching of the cardiac end of the stomach from the gastro-esophageal vestibule. The controver-

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sial criteria for diagnosing the smaller hiatal hernia led us to divide them into three degrees: First degree consists of herniations of the gastro-esophageal vestibule alone. Second degree hernias consist of those of gastro-esophageal vestibule and two or three centimeters of the cardiac end of the stomach, and in hernias of the third degree, at least four of five centimeters of the stomach lie above the diaphragm."

The exacting criteria adopted by these investigators would account for the higher incidence than reported by others. Their series is relatively small and the patients had some form of disturbance which necessitated x-ray studies.

I am also of the opinion that the incidence of true hiatus hernia, including all age groups, is higher than generally reported in the literature, most likely between 25% and 30% if the patients are carefully studied according to the criteria of the above authors.

Putney[5] in a paper presented in 1948 before this Society, called attention to the symptoms associated with this condition, and stressed the importance of differential diagnosis by endoscopic investigation between true hiatus hernia, so-called congenitally short esophagus and peptic esophagitis. The same author,[6] in another paper, discussed the mechanism of the esophago-gastric sphincter. Soulas,[7,8] in his monumental work on the esophagus, has also contributed to this subject.

ANATOMICAL AND EMBRYOLOGICAL CONSIDERATIONS

According to Terracol,[9] "There is no muscular structure in this region, whether it be the diaphragm itself or the tenuous fibers of Junara and Rouget which can play the role of a sphincter. There is absolutely no resemblance of any of these muscular structures to the cricopharyngeal mechanism, the fibers of which are an integral part of the wall of the esophagus and which contract with it. In fact, from the physiological point of view, the diaphragmatic canal in which the esophagus lies is not a true sphincter because it does not provide intrinsic muscle fibers comparable to those at the mouth of the esophagus."

If this is true, one can better understand the complications of esophagitis which may develop because of the abnormal relation between the stomach and the esophagus, allowing unimpeded regurgitation of acid and pepsin into the esophagus. The nature of the mechanism of food passage through the lower esophagus has been the

subject of much discussion, centered chiefly around the fact that material is held back at a point about two to three centimeters above the cardiac orifice of the stomach, as if there were a sphincter at that point. Various theories have been advanced for this, such as the influence of respiration, the constricting of the diaphragm at the esophageal hiatus, or the actual presence of intrinsic sphincter muscle.

None of these appear convincing. If there is a cardiac sphincter it cannot be very effective. In fact, free regurgitation of material from the stomach into the lower esophagus can usually be demonstrated in every normal subject, especially in the Trendelenberg position. The only reasonable explanation of the hesitation in the passage of material at this level is the sphincter-like tonicity of the smooth muscles of the esophageal wall, aided perhaps by a few striated fibers often present.

The behavior of the esophagus at its lower end, and of the cardia, is of special interest. Although no true sphincter or external-like mechanism can be demonstrated, the physiologic activity of the muscle layers of this portion serves to control, to some extent, the passage of material into the stomach. As the peristaltic wave reaches this area where the phenomenon referred to by the roentgenologists as the "phrenic ampulla" occurs, there is a peak rise in pressure with a plateau falling off to normal, similar to that which prevails in the main portion of the esophagus.

In the lower segment of the esophagus, corresponding to the portion in and below the esophageal hiatus of the diaphragm, the peristaltic activity is distinctly different. Here there is no forceful peristaltic wave but merely a gradual rise and fall of pressure, varying with the accumulation and subsequent passage of esophageal contents. This activity seems dependent upon intrinsic reflex peristalsis and appears to be characteristic of this segment only. Although locally independent of the activity of the upper portion of the esophagus, the action of this segment is integrated with it, because, although normally in a state of tonicity it relaxes as the main peristaltic wave reaches it from above.

These observations, based upon recent studies of intraluminal pressures during deglutition, help to explain the phenomenon observed on fluoroscopy, particularly of the distensible segment just above the diaphragm called the "phrenic ampulla." This would seem to correspond to the region in which the peristaltic waves are less forceful or absent. Very often, even though watching the passage of the barium

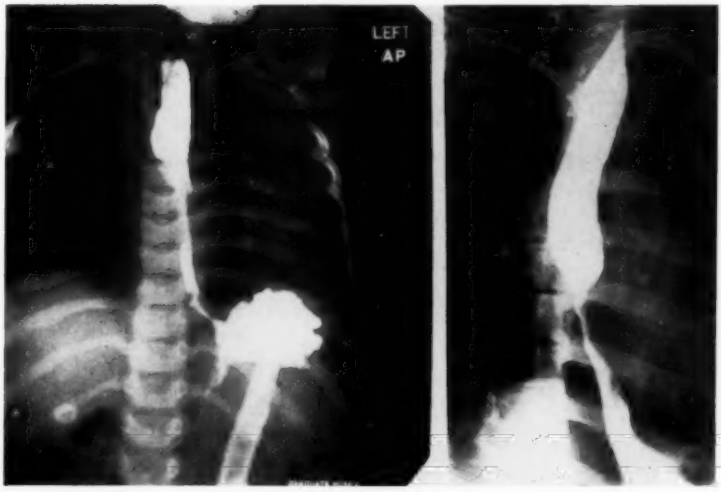

Figure 1

meal bolus carefully, one cannot always distinguish the portion of the esophagus between this segment and the cardia.

Cannon,[10] many years ago, showed that the region of the esophago-gastric segment responded very readily to changes in pH. Thus, in the presence of alkaline or neutral media, rhythmic relaxation occurs which permits regurgitation from the stomach into the lower esophagus and conversely, if the gastrict contents are normally acid this regurgitation ceases after a short latent period. The unimpeded regurgitation of acid and pepsin into the esophagus because of abnormal relation of the stomach and esophagus had led to varying degrees of esophagitis. This occurrence of esophagitis has given rise to the contention that the shortening of the esophagus is actually an acquired condition resulting from cicatricial contraction secondary to long-standing irritation.

One may concede that a small degree of shortening can develop in this manner; but it is highly improbable that this mechanism can be responsible for the upward displacement of the cardia to levels often as high as the aortic arch. It is a matter of common experience that the contraction of scar tissue in any tubular organ takes place much more in a circumferential than in a lengthwise direction in relation

to the long axis of the organ. This should certainly eliminate some of the confusion usually found in differentiating these two conditions. Roentgen examination of the esophagus has shown that short esophagus with a thoracic segment of stomach is not an uncommon occurrence in children and even among adults who have never had symptoms in early life.

The existence of this condition has been recognized in recent years by the characteristic x-ray changes of an abnormal lower segment of the esophagus and by subsequent esophagoscopic examination with histological confirmation of the presence of gastric mucosa in the area normally occupied by the esophagus.

In spite of the fact that there is this marked difference between hiatus hernia and true congenitally short esophagus, many clinicians are of the opinion that congenitally short esophagus is practically nonexistent and that it may be nothing more than an accentuation or variation of the usual type of hiatus hernia.

Congenitally short esophagus represents an embryological agenesis of the subtracheal segment of the esophagus which has been replaced by a fixed segment of stomach in the thorax. Recognition of this embryological anomaly reconciles the clinical findings associated with congenitally short esophagus. In most of these cases the abrupt change from the normal cylindrical contour of the true esophagus to that of the varied type configurations, ranging from ring-like stricture formation to marked dilatation of the entire lower segment normally occupied by the esophagus, further differentiates it from hiatus hernia.

CLINICAL OBSERVATIONS

Knowledge gained from over 5,000 esophagoscopic examinations over a period of 25 years has yielded considerable practical information in reference to the triangle of hiatus hernia, congenitally short esophagus and esophagitis.

Analysis of this information reveals that the incidence of hiatus hernia varies with the different decades, that it is less than 2% in the first five years of life from which time it gradually increases to 5% at fifteen years of age and by the fourth and fifth decade it occurs in 20% to 25% of people if properly examined.

On the other hand, the incidence of true congenitally short esophagus is less than 4% and at least 50% of the individuals thus afflicted develop abnormal esophageal symptoms so that the condition becomes

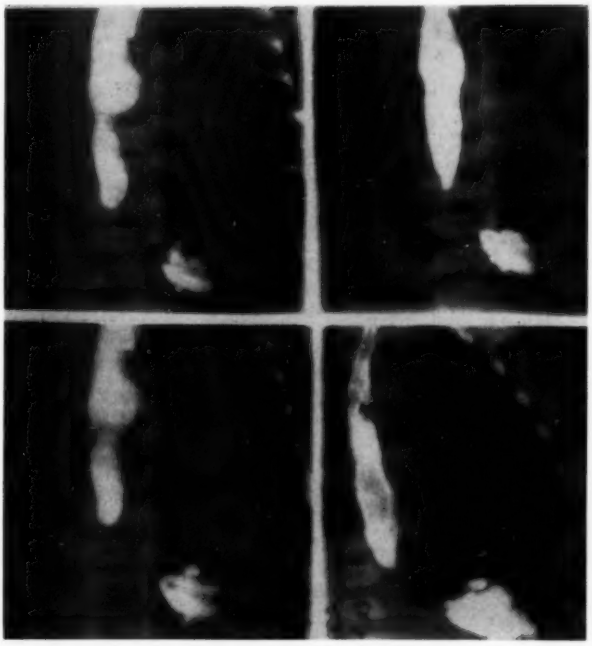

Figure 2

apparent either shortly after birth or in the first five years of life. Another 25% develop symptoms before the second decade, and the remaining 25% may not develop symptoms until much later in life. Direct interview with many of the parents of children with congenitally short esophagus revealed the predominant problem to be the persistent regurgitation of food with associated malnutrition. In several children in whom the severe symptoms of regurgitation did not develop until the second and third years of life, the diagnosis of "temper tantrums" was made until the true nature of the underlying abnormality was established.

REPORT OF CASES

The following case report clearly demonstrates the above points:

CASE 1. J.M., white, four years of age, was admitted to Graduate Hospital on July 21, 1937, with the chief complaint of being

unable to eat since birth. There had been persistent vomiting of food and liquids. Until five weeks prior he had been able to get some liquids down by repeated efforts. Several days prior he began to have increased difficulty, so that not even water would go down. Prior to this episode of complete obstruction, vomiting had not been projectile and regurgitated food was unchanged. The child had been admitted previously to another hospital and the conclusion was that the patient was normal organically and that the vomiting was willful.

X-ray studies made on present admission, however, showed that this child had a condition suggestive of congenitally short esophagus (Fig. 1). Since this child required a gastrostomy for nutrition, it was possible to examine the lower end of the esophagus below the point of constriction by retrograde esophagoscopy. Gastric mucosa was observed throughout the entire lower segment of the esophagus. This child was successfully dilated by retrograde bouginage for several years so that his gastrostomy was closed and he was subsequently treated with mercury bouginage.

Why the onset of symptoms is so variable may depend on the amount of stomach present in the thorax or the activity of the secretory cells and the effect upon the adjacent esophageal mucosa. Naturally, the more secretory activity, the more the irritative reaction. Unfortunately the above factors have not been given the consideration they properly deserve in understanding the varied types of complications associated with congenitally short esophagus.

Recently I had the privilege of discussing this problem with Dr. John W. Hope, the roentgenologist at Children's Hospital, and in the process of reviewing some of his interesting cases, we came upon the radiological studies of an infant that had been x-rayed at five, nine and seventeen days of age respectively, because of vomiting blood since birth. Each one of these individual studies is a revelation as to how one could come to an erroneous diagnosis on the basis of interpreting the findings of the studies individually (Figs. 2, 3 and 4).

In the films made on the fifth day, iodized oil was used as the contrast media instead of barium. In the remaining studies a barium mixture was used. Reviewing the films made on the fifth day, one could justly conclude that the most likely diagnosis might be cardiospasm. However, study of the films made on the ninth day reveal the characteristic changes associated with hiatus hernia. The films made on the seventeenth day reveal a pouch filled with barium, extending up to the level of the third dorsal vertebra, which has all the character-

Figure 3

istics of a segment or stomach, associated with a congenitally short esophagus. This interpretation was further substantiated by the fact that the infant also had inversion of the stomach, which occurs commonly with congenitally short esophagus. (This does not necessarily reflect Dr. Hope's personal opinion.)

In contrast to the patients who have symptoms immediately after birth, there are some who do not develop any severe discomfort until the fourth and fifth decade of life. However, a careful investigation into the past history of these patients reveals that they had varied types of distress ranging from mild heartburn to a raw, scalding sensation when food passed over a certain area of the esophagus. Many patients will even localize the distress to the lower half of the chest. Unfortunately, because of the misunderstanding of the underlying cause of these patients' difficulty, they have been subjected to many operations and the removal of organs that had no direct bearing on their symptoms. Thus many patients have had cholecystectomy, partial gastric resection, gastro-enterostomy and repair of a hiatus hernia

Figure 4

without any relief of their symptoms, simply because their symptoms originated in the abnormal segment of the stomach in the thorax and not in the organs removed.

A brief resumé of the following case will illustrate the pitfalls associated with this condition.

CASE 2. L.P., a white female, 65 years of age, had difficulty in swallowing and burning sensation in the lower half of chest while eating.

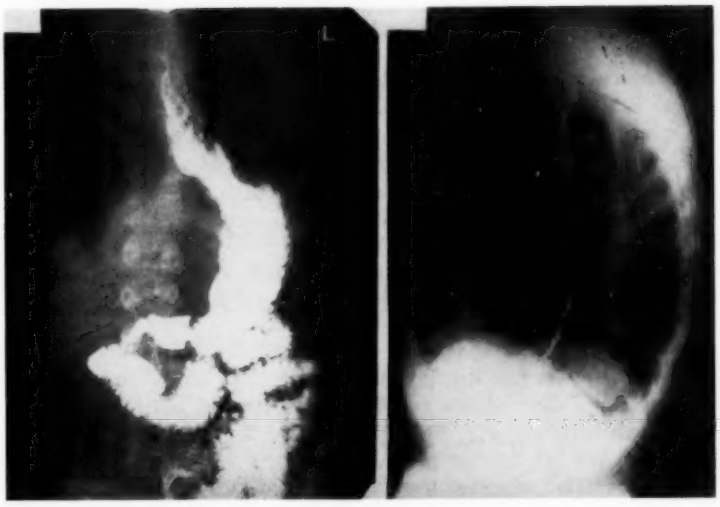

Figure 5

Patient recalled having had distress, described as an uncomfortable feeling on eating, most of her life. Certain foods such as "spicy or seasoned" left a raw sensation along the lower portion of her esophagus. In the last ten years her discomfort had become progressively worse. Following an episode of pain between her shoulders and abdominal distress, her gall-bladder was removed with no relief of discomfort. Exploratory laparotomy for suspected duodenal ulcer was performed but no ulcer was found. Another operation was performed for suspected gastric lesion, but nothing was found. The patient, by this time, was considered to be neurotic.

On her most recent admission to the hospital, because of inability to swallow even liquids, x-ray studies of the esophagus by Dr. Edwin L. Lame at Presbyterian Hospital on September 25, 1959 (Fig. 5), were as follows:

"Barium study of the esophagus reveals a sharp constriction at the transverse aorta and extending for three inches down, at which point the esophagus meets the upper border of the stomach. The stomach is above the diaphragm for a distance of about five inches and occupies an elongated course here. We do not believe the con-

striction is due to tumor. The constriction is constant and no doubt is a factor in the patient's difficulty in swallowing."

On esophagoscopic examination one noted a granular appearance of the mucosa at the level of the esophagus where the stenosis began. By dilating the constricted orifice it was possible to pass beyond this point, where it became apparent that there was an entirely different appearance of the mucosal surface. Now the appearance was typically that of rugal folds, which one sees in the stomach. Biopsy of this area revealed gastric mucosa with marked inflammatory changes.

Since gastric mucosa in this patient was found just below the level of the aortic arch, I am sure no one would doubt that this represented stomach at this high level.

One could report many other similar cases where the diagnosis of congenitally short esophagus was never suspected, or often confused with hiatus hernia.

SUMMARY

The thorough understanding of the difference between congenitally short esophagus and hiatus hernia is important because the treatment of these two conditions is different.

True hiatus hernia can be treated surgically with a good prognosis for relief of the patient's distress. No such result can be obtained by the same surgical procedure in the presence of congenitally short esophagus. In this condition the defect is not due to weakness of the diaphragmatic opening which allows variable parts of the abdominal stomach to slip through it, but rather to a congenital defect of embryological origin, whereby a portion of the stomach remained in the chest and replaced the lower portion of the esophagus. In true congenitally short esophagus no alteration occurs in the position of this segment of stomach whether in the upright or Trendelenberg position. This immediately distinguishes it from hiatus hernia.

Since the lower end of the esophagus is completely replaced by gastric mucosa it might avoid confusion in the future if a descriptive terminology were used to identify this condition, instead of congenitally short esophagus. I believe that the term gastrosa (gastric mucosa) of the esophagus would be indicative of the true nature of the anatomical structure in this altered condition. This should reconcile the differences of opinion so prevalent today in differentiating between hiatus hernia and congenitally short esophagus.

Since gastrosa of the esophagus occurs more frequently than suspected all efforts should be utilized to distinguish it from hiatus hernia.

The esophagitis which is practically always associated with gastrosa of the esophagus and frequently with hiatus hernia, may be treated more intelligently if the mechanism underlying its cause is clearly understood. Critical discussion among clinicians, roentgenologists, and esophagologists may lead to a better understanding of the interrelated problems of gastrosa of the esophagus, hiatus hernia and the associated esophagitis.

CONCLUSION

The differential diagnosis between hiatus hernia and congenitally short esophagus is important, since each represents an entirely different condition.

Congenitally short esophagus is the result of an embryological defect whereby a segment of esophagus is replaced by stomach. This is always present at birth.

Hiatus hernia is a defect in the opening of the diaphragm through which the esophagus passes into the stomach. This weakness of the diaphragmatic ring may be present at birth, but in 95% of the cases it is an acquired condition, the incidence of which increases with the later decades of life.

The treatment of these conditions is entirely different. The confusion of congenitally short esophagus with hiatus hernia may lead to operative procedures which will not contribute to the amelioration of the patient's symptoms and in fact might make them worse.

Since congenitally short esophagus is an embryological defect whereby a portion of thoracic esophagus is permanently replaced by stomach before birth, it might avoid further confusion in the future if it were designated by a descriptive term, namely: gastrosa (gastric mucosa) of the esophagus.

Peptic esophagitis in varying degrees is practically always associated with gastrosa of the esophagus and may or may not be a complicating factor in hiatus hernia.

Bleeding is a more frequent complication of gastrosa of the esophagus than hiatus hernia.

The alert roentgenologist has contributed valuable aid in the differential diagnosis of disorders of the esophagus. The well-trained broncho-esophagologist should be of further aid in the differential evaluation of these conditions.

The gastro-enterologist, by his close observation of these patients, will be helpful in outlining the type of therapy best suited for each individual case.

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REFERENCES

1. Kirklin, B. R., and Hodgson, J. R.: Roentgenologic Characteristic of Diaphragmatic Hernia. Am. Jour. Roentgenology 58-77, 1947.

2. Nuzum, R.: Hernia of Esophageal Hiatus, Its Relation to Angina Pectoris. Am. Heart J. 33:724-, 1947.

3. Olsen, Arthur M.: Tr. Am. Broncho-Esophagological Association, p. 96, 1948.

4. Stein, George N., and Finkelstein, Arthur: In press, Graduate Hospital, Philadelphia, Pa.

5. Putney, F. Johnson: Tr. Am. Broncho-Esophagological Association, p. 85, 1948.

6. Putney, F. Johnson: Esophagastric Sphincter Incompetency. Tr. Am. Laryng., Rhinol. and Otolog. Society, p. 102, 1959.

7. Soulas, A.: Spasmes et Dyskinesies de L'oesophage. Encyclopedia Medico Chirurgicale, p. 1 (Mar.) 1954.

8. Soulas, A.: Oesophagite Ulcereuse. Encyclopedie Medico Chirurgicale, p. 1 (Oct.) 1953.

9. Terracol, J.: Les Maladies de L'Oesophage. Masson et Cie, 1951.

10. Cannon, W. B.: Acid Closure of Cardia. Amer. J. Physiology 23:105-114, 1908.

LXXXVII

TREATMENT OF ESOPHAGEAL STRICTURES

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AND

GEORGE C. HENNIG, M.D.
(by invitation)

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There are many accepted methods for the treatment of benign strictures of the esophagus. It is to be expected that one method will not apply to all strictures. The instrumentarium that is available will vary in different clinics. The background and skill of the individual assigned to the treatment is also a factor in the choice of method.

For several years, due to my earlier training under Dr. Louis Clerf, I was of the opinion that no one should treat a stricture of the esophagus except a trained endoscopist. Blind bouginage was to be condemned because of the dreaded perforation that might occur. Richards and Dietrich[1] presented a paper on this subject before this association in 1952. They showed that if care is exercised, blind bouginage can be successfully carried out.

Treatment of esophageal strictures by means of bougies introduced through the esophagoscope is not too satisfactory. The degree of dilatation is limited by the internal diameter of the tube. A 9 mm esophagoscope will only accommodate a 26 bougie (French Scale). It is usually necessary to use a larger bougie or olive if a good result is to be obtained. In our clinic, we try to dilate strictures over a period of time up to 40 French and higher if possible.

Care is necessary in the dilatation of strictures. One complication that may occur is splitting of the esophagus[2] at the stricture level. This can occur if the treatment is too aggressive and too large a dilator is used. Fortunately, it is not too serious and the condition usually subsides without the need of surgery as compared with a perforation.

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Clerf[3] stated that he preferred using olive-tipped fenestrated bougies that are passed over a previously swallowed thread. Broyles[4] devised a technique that utilized the wire staff of a small esophageal bougie in order to guide metal dilators through a stricture. The bougie also acted as a stopper for the dilator. Hoover[5] demonstrated that it is possible to secure a greater dilatation of a stricture by introducing Jackson dilators, one at a time, through the esophagoscope and below the stricture. On withdrawing the dilators, he allows two, three, or even four bougies to rest in the stricture simultaneously.

Crump,[6] in 1928, described a method using a metal wire technique for the dilatation of strictures. Crump made his own instruments. His methods could not be used in other clinics because the instruments were not available. They are now being manufactured by G. P. Pilling and Son Co. Crump and Hennig[7] reported on the use of this technique in securing biopsies of esophageal ulcers by retrograde curettage. The method is described in detail in their article.

The principal instruments devised by Crump include a flexible tip wire, a flexible hollow carrier for the wire, a knob-tipped staff wire with a central opening through the knob, and a hollow staff carrier to which metal bougies with a central opening can be attached. Retrograde curettes can also be attached to the same staff carrier.

The technique for their use as described by Crump and Hennig is as follows: "With the patient in a sitting position and the head hyperextended, a hollow flexible metal carrier is passed to the site of the obstruction in the esophagus, and a flexible tipped wire is then introduced through the lumen of the carrier.

"1) When the wire reaches a point of resistance, the carrier is withdrawn a few centimeters.

"2) The flexible tipped wire is gently rotated until it finds the lumen, then gently advanced past the site of obstruction.

"3) The carrier can now also be advanced past the site of obstruction, and if further areas are encountered, the same steps are repeated. In this manner, the flexible tipped wire is advanced into the stomach.

"4) The carrier is withdrawn leaving the flexible tipped wire in place.

"5) A perforated knob-tipped wire is passed over the flexible wire into the stomach and the latter removed.

"6) The blunt knob-tipped wire now serves as a safe guide for other instruments preventing perforation of the esophagus. The knob-tip will not allow the other instruments to advance beyond the end of the wire.

"7) A hollow staff to which is attached a metal bougie is passed over the wire. Graduated sizes of the metal bougie are passed up to the desired dilatation."

The hollow staff and metal bougies can also be passed over a previously swallowed thread.

There are instances where the metal guide wire will not find the lumen of the stenotic area. The tip of the wire is flexible and is referred to, in the clinic, as a cat's whisker, it will bend rather than penetrate or perforate the mucosa. In the event that a lumen is not found, an esophagoscopy is performed and the wire is passed under direct vision. After the wire passes beyond the obstruction, the esophagoscope is withdrawn and the same steps as outlined above are followed.

There are occasions where neither a wire or string will pass beyond a strictured area. In such cases, other methods for dealing with the stricture are necessary.

The Crump instruments have certain advantages over other equipment. The wire makes a firm guide for the metal bougies. The latter, because the perforation is centrally located, allows for an equal amount of force against the wall of the strictured area. The procedure can be used on outpatients without doing an esophagoscopy. The method has none of the disadvantages of a string. The latter can become tangled. Also, it may take one or more days for the string to pass through the bowel, which can be a nuisance.

Included in the Crump instruments are retrograde curettes for securing biopsies. He secured a biopsy in 90% of cases in which it was used. Many of these cases had a previous esophagoscopy with a negative biopsy.

Retrograde bouginage can be carried out through a gastrostomy opening by attaching the metal olives to a previously swallowed string.

The method has proved to be safe. It overcomes many of the objections of blind bouginage in that the flexible tipped wire cannot

perforate. Once the knob-tipped wire is in place, dilatation with metal olives is easily accomplished without too much discomfort to the patient.

SUMMARY

The Crump method of treating strictures of the esophagus has been described.

The technique has been found to be a safe and effective form of therapy.

The advantages of this method have been discussed.

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REFERENCES

1. Richards, L., and Dietrich, H. J.: Blind Bouginage in the Treatment of Benign Esophageal Obstruction. Annals of Otology, Rhinology and Laryngology 61:1134, 1952.

2. Jackson, C., and Jackson, C. L.: Bronchoesophagology. W. B. Saunders Company, 1950.

3. Clerf, L. H.: Discussion of paper by Richards and Dietrich. Trans. Amer. Broncho-Esophagol. Assn., p. 52, 1952.

4. Broyles, E. N.: Personal Communication.

5. Hoover, W. B.: Esophagitis: A Clinical Evaluation. Annals of Otology, Rhinology and Laryngology 61:1148, 1952.

6. Crump, A. B.: Diseases of the Esophagus. Blumer's Bedside Diagnosis, Vol. 1,650. W. B. Saunders Co., 1928.

7. Crump, A. B., and Hennig, G. C.: The Diagnosis of Carcinoma of the Esophagus: The Method of Biopsy with the Retrograde Curette. Gastroenterology 13:153, 1949.

LXXXVIII

OCCULT BRONCHOGENIC CARCINOMA

CASE REPORT

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The successful application of precision bronchoscopy in the early diagnosis of bronchogenic carcinoma has been noted by numerous authors. This diagnostic aid has become a procedure of prime importance in detecting bronchogenic carcinoma in the curable stages. With the development of successful thoracic surgical techniques it has become increasingly important to diagnose the lesion during the early stage of bronchial irritation rather than in the later advanced stages of bronchial obstruction. Since the lesion is usually located in close association to the main bronchial divisions, it lends itself to early diagnosis by precision bronchoscopy.

It would be of considerable clinical importance to detect bronchogenic carcinoma in a patient with a normal admission chest film. It would further arouse diagnostic interest to detect bronchogenic carcinoma in a patient with a normal bronchogram. The interesting aspect of this case report is that both of these negative roentgenographic studies were obtained from an individual with bronchogenic carcinoma proven by bronchoscopic biopsy. Since the patient's only symptom was mild nonproductive cough, this diagnosis certainly represents detection in the potentially curable stage of bronchial irritation.

The patient was a 67 year old white male who was admitted to the Minneapolis Veterans' Hospital because of cramping abdominal pain of six weeks' duration. A gastrointestinal x-ray study was ordered. Because of a history of chronic nonproductive cough, the astute internist also ordered routine studies of the sputum for tubercle bacilli, fungi, and tumor cells. The third sputum specimen was reported as suspicious for tumor cells. As a result of these studies a complete pulmonary work-up was initiated.

1. Posterior-anterior and lateral films of the chest — normal
2. Planograms of the right lung field — normal

3. Planograms of the left lung field	—	normal
4. Bronchoscopy and left bronchogram	—	normal
5. Right bronchogram	—	normal
6. Planograms of the mediastinum	—	normal

Nine additional sputa were collected and one appeared more definitely suggestive of malignancy. As a result of this unusual situation bronchoscopy was again requested three weeks later. A complete evaluation of the upper and lower respiratory tracts was undertaken. The nasopharynx, oropharynx, hypopharynx, vocal cords, trachea, carina, right main stem bronchus and its subdivisions were examined and reported to be normal. While carefully examining the subdivisions of the left upper lobe a definitely invasive lesion 5 mm in diameter was noted inside the lower lip of the left upper lobe orifice. The same bronchoscopist had reported this mucosal area as being within normal limits three weeks previously. This lesion undoubtedly represented an early mucosal breakthrough too minute to have been recognized at the earlier examination.

Biopsy was accomplished with the Holinger upper lobe resection telescope, the flexible arm being directed laterally until the area of the lesion was reached. A direct mucosal bite was taken. The pathological finding was squamous cell carcinoma.

The patient underwent a left radical pneumonectomy. The pathologist could not demonstrate gross evidence of tumor in the resected specimen. Microscopic section of the perihilar lymph nodes showed no evidence of tumor. Serial sections of the site of previous biopsy showed early submucosal extension of a squamous cell carcinoma surrounded by islands of carcinoma in situ.

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REFERENCES

1. Putney, J. F.: Bronchoesophagology. A.M.A. Archives of Otolaryngology 69:620-646 (May) 1959.

2. Goldman, J. F., Freeman, J.: Carcinoma of the Bronchus with Negative X-ray Findings. Annals of Otology, Rhinology and Laryngology 63:500-508 (June) 1954.

3. Looper, E. A., Cross, P. J.: Early Diagnosis of Bronchogenic Carcinoma. Annals of Otology, Rhinology and Laryngology 58:1113-1123 (Dec.) 1949.

4. Eeman, Fernand: Endoscopic Diagnosis of Early Bronchogenic Carcinoma. Journal of Laryngology and Otology 67:266-276 (May) 1953.

5. Umiker, William: Cytology in Bronchogenic Carcinoma. Amer. Jour. of Clin. Path. 22:558-563 (June) 1953.

6. Ochsner, A., DeCamp, P. T., Roy, C. J.: Bronchogenic Carcinoma with Emphasis on Early Diagnosis. Geriatrics 9:15-19 (Jan.) 1954.

7. Boucot, K.: Preclinical Bronchogenic Carcinoma. Amer. Jour. of Surg. 89: 457-461 (Feb.) 1955.

8. Jewett, J. S.: The Early Recognition of Bronchogenic Carcinoma. Dis. of Chest 22:699-708 (Dec.) 1952.

9. Overholt, R. H., Woods, F. M.: Early Diagnosis and Treatment of Carcinoma of the Lung. New Eng. Jour. of Med. 245:555-560 (Oct.) 1951.

10. Rigler, L. G.: Roentgen Examination of the Chest. J.A.M.A. 142:773-777 (Mar.) 1950.

LXXXIX

MICROSCOPIC SUSPENSION LARYNGOSCOPY

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HAROLD G. TABB, M.D.
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The suspension laryngoscope introduced by Killian in 1909 was a crude instrument, but inspired others to modify and improve it. The modifications devised by Lynch in 1914, along with others which he added over the next few years, converted an initially crude apparatus into an instrument that led to remarkable advances and refinements in intralaryngeal surgery.[1] So well conceived were Lynch's modifications, in fact, that the suspension laryngoscope in use today is essentially the instrument that he described in 1914. In the intervening years, the indications for, contra-indications to, and technique of, suspension laryngoscopy have been definitively described, first by Dr. Lynch himself and later by his son, the late Dr. Mercer G. Lynch, and their associate of many years, Dr. Francis E. LeJeune.[2]

This presentation is limited to a description of microscopic suspension laryngoscopy, a modification of the Lynch technique that we have employed for the last year. It permits even greater refinements in intralaryngeal surgery than are possible with the original technique. It also permits more precise and more delicate surgery because the use of a microscope with the suspension laryngoscope enlarges and brilliantly visualizes the smallest lesion in the area in three-dimensional proportions. It is not an exaggeration to say that a pea-sized lesion, when observed through the microscope, seems almost the size of an orange.

These results are achieved by the addition to the Lynch suspension laryngoscope of a binocular operating microscope. All our experience has been with the Zeiss instrument used for surgery on the ear, though, conceivably, other microscopes might be adapted for this

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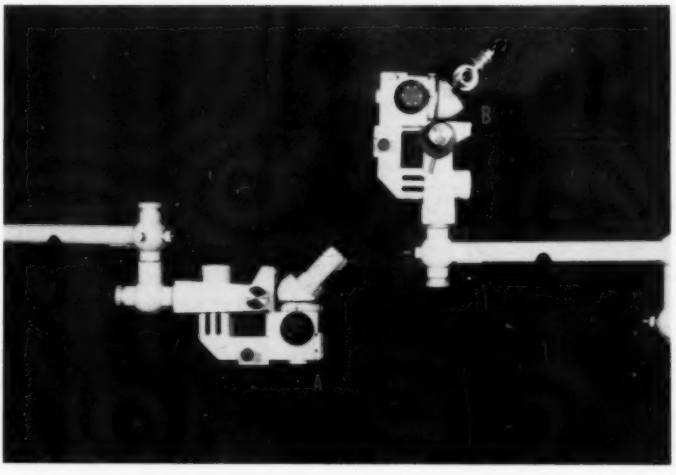

Fig. 1.—Left, Zeiss binocular microscope for otologic surgery. Right, microscope adapted for suspension laryngoscopy.

purpose. Whatever microscope is used must meet two specifications: 1) It must have angulated eye pieces. Straight eye pieces are not satisfactory for intralaryngeal surgery. 2) Even more important, the microscope must have a 300-mm objective. Any working distance less than 12 inches is not practical.

It is a simple matter to adapt the microscope used in ear surgery (Fig. 1A) for use in suspension laryngoscopy. The first step is to remove the short arm between the horizontal bar and the lens system. The second step is to replace the lens system, not in its original position (Fig. 1A), but on the superior aspect of the horizontal bar (Fig. 1B).

Pre-operative preparation, anesthesia, and surgical technique in general conform to standard practices. Pre-operative preparation consists of Seconal sodium (sodium propylmethylcarbinylallyl-barbiturate) the night before operation and Demerol (meperidine hydrochloride) and atropine on call to the operating room. The average adult dosages are used, that is, 100 mg of Seconal, 100 mg of Demerol, and .4 mg of atropine.

The mouth and laryngopharynx are lightly sprayed with 1 cc of 4 per cent cocaine. The larynx is then blocked by means of laryngeal

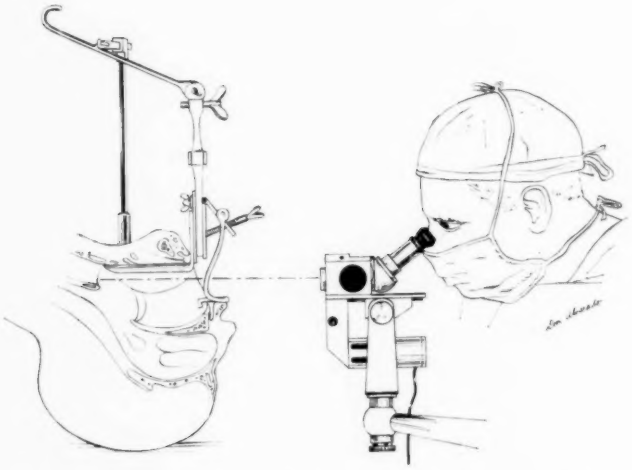

Fig. 2.—Details of placement of microscope and Lynch suspension laryngoscope. Line of vision (indicated by dotted line) from microscope to upper teeth and thence to base of epiglottis.

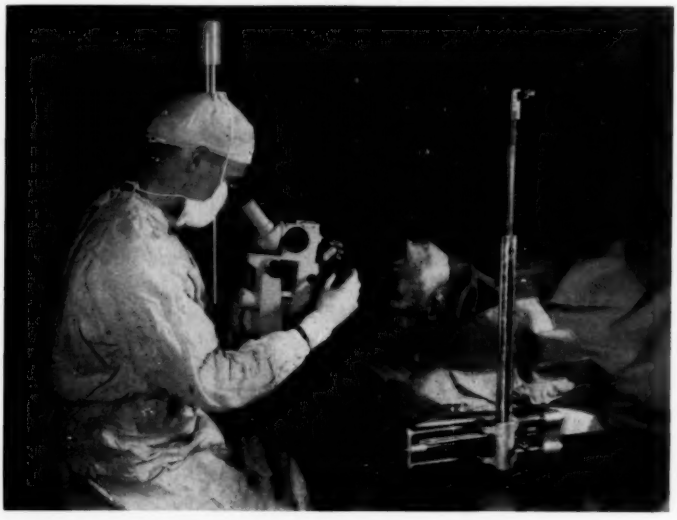

Fig. 3.—Removal of intralaryngeal nodule by the suspension laryngoscopic technique, with the microscope.

cotton applicators, and by a supplemental transcricoid injection of 1.5 to 2 cc of 4 per cent cocaine. We never exceed a dosage of 5 cc of cocaine.

Suspension laryngoscopy requires deep anesthesia, which implies the services of a highly skilled anesthetist. Anesthesia is achieved with Pentothal sodium (Thiopental sodium). When the point of unconsciousness is reached, curare is used to relax the muscles of the mandible and tongue. These are both potentially dangerous drugs and must be used with skill and caution. During the entire procedure, oxygen is fed into the oral cavity per cannula, and an anesthetic machine is kept immediately at hand for use if artificial respiration should become necessary.

The upper teeth are protected with the hard rubber mold (gump) now available in both adults' and children's sizes instead of with the lead plate previously used. The mouth is held open with a side gag.

With the aid of a Lempert headlight or some similar light, the Lynch suspension apparatus is placed in position (Fig. 2), so that the spatula engages and elevates the laryngeal surface of the epiglottis. When this has been accomplished, the vocal cords are well visualized. It is important that the spatula be centered in the midline, to avoid trauma to the tongue and the tonsillar pillars.

The appropriate hook of the suspension apparatus is then attached to the crane (Fig. 2), which is gently elevated and which elevates, in turn, the tongue and soft tissues, with resultant direct visualization of the larynx.

If exposure is not sufficient with the first manipulations, the crane is adjusted until maximum visualization is obtained. It is essential that the surgeon be able to look along a straight line from the upper teeth to the base of the epiglottis and that the light beam from the microscope (Fig. 2) be part of the same straight line. If a straight line from the teeth to the epiglottis cannot be accomplished, suspension laryngoscopy is unsuited for the special case and surgery by this technique must be abandoned. All laryngologists have encountered cases of this sort, and the situation is not altered when the microscopic technique is employed.

When the suspension apparatus has been placed satisfactorily and visualiation of the entire larynx, laryngopharynx and pyriform sinuses has been accomplished, the surgeon removes his headlight and moves

the microscope into position. Once it is adjusted (Fig. 3), his hands are left free for any required surgery upon the greatly magnified vocal cords.

Up to the present time, we have used standard instruments for laryngeal surgery by this technique, but we do not find them satisfactory. The enlargement achieved by the use of the microscope affects instruments as well as lesions, and the surgeon often feels as if he were working with crowbars. We are now working on designs for more delicate instruments.

We have found this technique particularly applicable to the removal of small, benign lesions confined to the true vocal cords, including polyps, papillomas, leukoplakic lesions, and inclusion cysts. By this method, it is possible to approach the larynx with the same precise, delicate techniques now employed in stapes surgery. Because the field of operation is so greatly magnified, only tissue that is clearly pathologic need be removed, and the resultant decrease in trauma is associated with a highly desirable increase in the rate of postoperative healing.

SUMMARY

The use of a Zeiss binocular microscope, designed for otologic surgery, with the Lynch suspension laryngoscope has made possible more precise and more delicate surgery upon the vocal cords and has, at the same time, reduced trauma and enhanced the rate of healing.

REFERENCES

1. Lynch, Robert C.: New Technic for the Removal of Intrinsic Growths of the Larynx. Laryngoscope 24:645-657 (July) 1914.

2. LeJeune, Francis E., and Lynch, Mercer G.: Laryngoscopy. In Otolaryngology (ed. George M. Coates, Harry P. Schenck, and M. Valentine Miller). W. F. Prior Company, Inc., Hagerstown, 1957. Vol. 4, chapter 25, pp. 1-10.

XC

ESOPHAGEAL DEFECTS IN DERMATOMYOSITIS

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Dermatomyositis is a relatively rare, nonsuppurative, nonhemorrhagic type of polymyositis in which cutaneous changes usually occur. In some instances involvement of the skin may be transient or completely absent, in which case the term "polymyositis" is applied. The primary pathologic change occurring in muscle is an inflammatory reaction followed by granulation and hyaline degeneration. The pathology of the skin lesions is nonspecific. Scleroderma, with edema and Raynaud's changes, and systemic lupus erytheratosus, with chronic skin lesions, clinically may simulate forms of dermatomyositis and give rise to such terms as "sclerodermatomyositis" and "poikilodermatomyositis" respectively. Dermatomyositis and scleroderma may simulate other diseases involving the esophagus and must be differentiated from such conditions as central nervous system disease, malignant tumors and achalasia, as well as from each other. In the absence of well-defined skin lesions, the clinical diagnosis may be obscured by the diffuse symptomatology and nutritional deficiency.

Dysphagia occurs in most of the so-called collagen diseases but it is encountered most frequently in dermatomyositis. In a review of 270 cases of dermatomyositis reported by Christianson and associates[1] more than 60 per cent of the patients complained of dysphagia. The medical literature on dermatomyositis contains few references to esophageal involvement, other than to mention the symptom, dysphagia.[2-4] To determine the esophageal involvement in dermatomyositis, the records of the Mayo Clinic were reviewed and 38 patients were selected in whom a diagnosis of either dermatomyositis or polymyositis had been made, and in whom investigation of the esophagus, either by roentgenography, esophagoscopy, and motility studies or by

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each of these methods, had been performed. Approximately half of the patients were selected from the presteroid era of therapy.

Dysphagia occurred in 32 of the 38 patients. Two of the 32 patients were unable to swallow even thin barium. With the sole exception of the patient having an esophageal hiatal hernia with stricture, the patients localized the sensation of obstruction to the pharyngeal area. The patient with stricture in the lower portion of the esophagus indicated that the sensation of obstruction occurred beneath the lower part of the sternum. Solids were swallowed with greater difficulty than liquids in all patients having dysphagia, but 11 patients noted intermittent regurgitation of liquids through the nose. In most patients the severity of the dysphagia was variable and was concurrent with progressive, generalized muscle weakness. When remissions of the disease occurred, complete remissions of dysphagia also occurred; such a favorable course was especially apparent among the group of patients maintained on adequate amounts of steroids. Most patients also noted dysarthria or a change in vocal tone during dysphagia.

Procedures utilized to establish a clinical diagnosis of dermatomyositis in the entire group reflected experience gained in evaluation of individual tests. Prior to 1956 the quantitative urinary excretion of creatine and creatinine was used as an index of muscle degeneration; 15 of the 17 patients in whom this quantitative test was performed had increased urinary excretion of creatine. Because of inaccuracies inherent in the procedure since 1956 it has been supplanted by the test for glutamic oxalacetic transaminase in the serum. This enzymatic determination correlated well with activity of the disease and has been employed to estimate muscle destruction. In 15 of the 16 patients in whom this test was performed, amounts of serum glutamic oxalacetic transaminase greater than the normal (1.43 micromoles per hour per milliliter) were present. In one patient in whom normal values were obtained, the level reflected large doses of steroid medication administered over three months prior to the test. The concentration of transaminase in the serum was observed to revert to normal in 75 per cent of the patients treated with adequate amounts of steroids for three weeks or longer.

Electromyographic studies were obtained in 32 patients. In 30 patients the tracings showed abnormal patterns that were considered compatible with the diagnosis of a myopathic disorder. In two patients the changes in the muscle fibers were minimal and not considered to be diagnostic. However, in both of these patients the concentrations of glutamic oxalacetic transaminase in the serum were elevated.

TABLE I

X-RAY EXAMINATION

FINDINGS		NUMBER OF CASES
Normal esophagus		19
Abnormal esophagus		16
Diminished peristalsis	9	
Absent peristalsis	3	
Laryngeal aspiration	3	
Diaphragmatic hernia	1	
Total		35

X-ray examinations of the upper gastrointestinal tract were made in 35 of the 38 cases; the results are compiled in Table I. As mentioned earlier, two patients were unable to swallow even thin barium; in a third patient x-ray examination was not attempted because of the risk of serious laryngotracheal aspiration of the barium. The esophagus was found to be normal in 19 patients; in nine, peristaltic activity was diminished and in three peristaltic activity was not demonstrated. Laryngotracheal aspiration of barium occurred in three patients. Only one patient was found to have an esophageal hiatal hernia which was complicated by stricture at the esophagogastric junction. In this patient, as well as in the 11 other patients having partial or complete motor failure, the appearance and response of the esophagus during roentgenoscopy were indistinguishable from the appearance of the esophagus in patients with scleroderma. In 12 patients in whom abnormal peristaltic activity was demonstrated, a diagnosis of scleroderma was suggested by the examining roentgenologists.

Esophagoscopy was performed in six patients. This reflects in part the absence of a specific diagnostic value of esophagoscopy in dermatomyositis and in part a reluctance to perform the procedure on an extremely weak, seriously ill patient. Esophagoscopic examination was considered necessary only in patients in whom a satisfactory x-ray examination could not be obtained or in whom the presence of an esophageal neoplasm could not be excluded. Esophagoscopic findings are tabulated in Table II. In four patients the entire esophagus appeared normal. In one the esophageal wall had lost its muscle tone. In the remaining patient the mucosa presented an atrophic

TABLE II

ESOPHAGOSCOPIC FINDINGS

CASE	ESOPHAGOSCOPIC REPORT
1	Normal
2	Normal
3	Normal
4	Normal
5	Atonic esophagus
6	Atrophic mucosa

appearance. Mucosal ulceration was not encountered and none of the patients had an esophageal hiatal hernia.

Esophageal motility studies were obtained in 17 patients. This method of study is a more sensitive indication of esophageal muscular function, and esophageal muscular derangements were detected by it more frequently than by roentgenograms. In only nine patients was a normal tracing obtained. Various types of motility derangements were found in the remaining eight patients and in none was the motor abnormality confined to the pharyngeal area alone. In four patients peristaltic contractures were feeble and inco-ordinated; three patients suffered from complete motor failure of the esophagus extending from a point 3 to 5 cm below the introitus to the gastroesophageal junction. One patient had a mild to moderate degree of diffuse spasm involving the lower third to half of the esophagus characterized by obliteration or replacement of the peristaltic wave with a simultaneous or prolonged contraction of the lower part of the esophagus. This patient with diffuse spasm and two of the patients with feeble peristalsis had an increase in the pressure barrier at the gastroesophageal junctional area. Results of the motility studies are tabulated in Table III.

COMMENT

The presence of a generalized esophageal muscular defect substantiated both by roentgenographic and motility studies in 45 per cent of the patients studied is noteworthy. With the exception of a patient in whom an esophageal hiatal hernia was found, the clinical history indicated that the sensation of dysphagia was localized to the

TABLE III

ESOPHAGEAL MOTILITY

FINDINGS		NUMBER OF CASES
Normal motility		9
Abnormal motility		8
Diminished peristalsis	4	
Absent peristalsis	3	
Diffuse spasm	1	
Total		17

pharyngeal area. Weakness of the pharyngeal muscles obscured symptoms of dysphagia arising in the esophagus. The esophageal defects in dermatomyositis, when measured by x-ray examination and motility studies, are sufficiently similar to the esophageal defects in scleroderma to be indistinguishable. One patient having motility studies was found to have intraluminal pressure changes on swallowing that were consistent with the pattern of diffuse spasm which was asymptomatic. Only two patients complained of lower substernal burning; one of these patients had a hiatal hernia with stricture, but the remaining patient did not have evidence of esophagitis at the time esophagoscopy was performed.

In this small series the incidence of hiatal hernia or evidence of ulcerative esophagitis complicating dermatomyositis was sufficiently infrequent to suggest the preservation of a protective mechanism in the esophagus. One of the mechanisms which prevents gastric reflux[5] and the development of esophagitis is an elevated esophageal pressure barrier above the gastroesophageal junction. This zone of elevated pressure was present in 65 per cent of the patients with dermatomyositis in whom motility studies were performed. Longer observation of these patients may reveal the development of a greater incidence of esophageal hiatal hernias, especially since three patients suffered total motor failure of the esophagus.

The principal clinical problem is in the differential diagnosis of dermatomyositis and scleroderma. The differences in the two diseases are sufficient to render diagnosis possible, yet the similarity of esophag-

TABLE IV

COMPARISON OF ESOPHAGEAL FINDINGS IN DERMATOMYOSITIS AND SCLERODERMA

FINDINGS	DERMATOMYOSITIS	SCLERODERMA
Clinical		
Dysphagia	Pharyngeal	Sternal
Nasal regurgitation	Frequent	Absent
Stage of disease	Severe muscle disease	With widespread surface lesions
Remission	With adequate steroids	None
Complications	Infrequent	Esophagitis, hernia, ulceration
Esophagoscopic	Not diagnostic	Hernia, esophagitis, ulceration
X-ray	Loss of peristalsis	Loss of peristalsis
Motility	Loss of motility	Loss of motility

eal involvement, particularly on the roentgenograms, is a frequent stumbling block to correct diagnosis. Table IV shows a comparison of clinical, laboratory and x-ray findings in dermatomyositis and scleroderma. More routine studies of patients with dermatomyositis and increased knowledge of the role of the esophagus in the disease should lead to greater clinical appreciation of this vital part of the picture of dermatomyositis. It should be noted that the laboratory procedures alone do not distinguish dermatomyositis and scleroderma; this is the province of the clinician and it is a judgment that can be made readily with present knowledge.

CONCLUSIONS

1. Dysphagia is a frequent symptom in dermatomyositis and is favorably affected by steroid therapy.

2. Although the patients localized the site of esophageal obstruction to the pharyngeal region, a generalized esophageal muscular involvement frequently was found.

3. The roentgenographic and esophageal motility patterns in dermatomyositis and scleroderma were indistinguishable.

4. The infrequence of ulcerative esophagitis and esophageal hiatal hernia in dermatomyositis suggests the preservation of a protective barrier mechanism in the lower portion of the esophagus.

MAYO CLINIC

REFERENCES

1. Christianson, H. B., Brunsting, L. A., and Perry, H. L.: Dermatomyositis: Unusual Features, Complications, and Treatment. A.M.A. Arch. Dermat. 74:581-589 (Dec.) 1956.

2. Sheard, Charles, Jr.: Dermatomyositis. A.M.A. Arch. Int. Med. 88:640-658 (Nov.) 1951.

3. Mattingly, S.: IV. Polyarteritis, Scleroderma, Dermatomyositis, and Thrombotic Thrombocytopenic Purpura. Ann. Phys. Med. 8:304-309 (Nov.) 1958.

4. Black, R. L., and Bunim, J. J.: Progressive Systemic Sclerosis (Scleroderma) and Dermatomyositis. M. Clin. North America (Nov.) pp. 1653-1662, 1958.

5. Fryke, F. E., Jr., Code, C. F., and Schlegel, J. F.: The Gastroesophageal Sphincter in Healthy Human Beings. Gastroenterologia 86:135-150, 1956.

XCI

RELATIONSHIP OF PULMONARY FUNCTION TESTING TO BRONCHOLOGY

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At the 24th Annual Meeting of the American Broncho-Esophagological Association in 1941 it was stated,[1] "A correlation between the bronchoscopic findings and the characteristics of respiration will, I believe, contribute to our knowledge of the pathological physiology and the treatment of certain obstructive diseases of the bronchi, especially bronchial asthma and hypertrophic pulmonary emphysema." This paper represents the continuation of that study and reports on a specific correlation that has been observed.

METHODS

The 9-liter Collins spirometer with low resistance valves, ventilograph, and two-speed drum is used. Examinations are performed at least one hour after eating, after a 20 minute rest period, and with the patient sitting up. A period of at least 5 minutes of quiet breathing is followed by a vital capacity and maximum breathing capacity determinations. The examination is performed first while breathing air with added oxygen to compensate for that absorbed during the examination, while breathing oxygen, and in many patients after such therapeutic procedures as the administration of a broncho-dilator aerosol and intermittent positive pressure breathing.

The vital capacity is recorded by the patient continuing a normal exhalation to the maximum extent, doing a fast maximum inspiration followed by a fast maximum exhalation.

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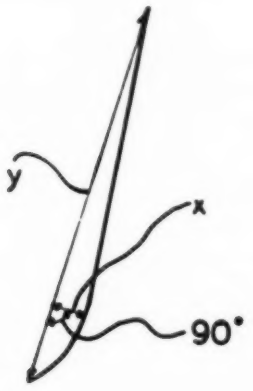


$$\text{Depth Index \%} = \frac{x}{y} \times 100$$

Fig. 1.—*Depth Index*: A means of expression of the expiratory vital capacity curve. Y is the distance between the beginning of the high speed flow and the end of the exhalation. X is the greatest distance at 90° to Y between Y and the vital capacity line.

$\frac{X}{Y} \times 100 =$ Depth Index in per cent.

Early in this study it was recognized that the increased expiratory curve showed the characteristic alteration in the vital capacity of patients with diffuse obstructive pulmonary emphysema. For the purpose of tabulation, a means of describing the curve was necessary. After reviewing the conventional mathematical and graphic methods, it was concluded that these were too cumbersome. An expression, the "Depth Index," was devised in order to attach a numerical value to the extent of the expiratory curve. This expression is not a completely accurate description of the curve, however, studies on normal subjects have shown values under 4 per cent; patients with significant functional impairment of the types associated with bronchial asthma or obstructive pulmonary emphysema have shown values over 4 per cent.

Maximum speeds of inspiration and expiration are determined by laying a straight edge along the most vertical portions of the record for a distance equivalent to at least 300 cc, drawing a line parallel to this, and determining the speed of air movement expressed in cubic milliliters or liters per second. Drum speed is 4.7 mm per second.

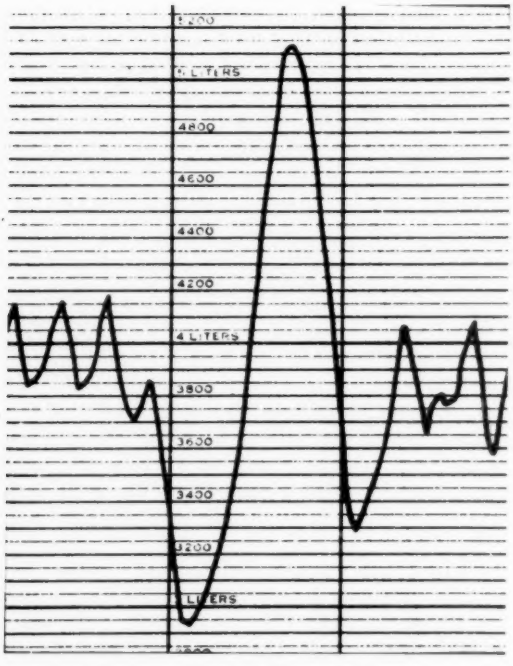


Fig. 2.—*Vital Capacity: Bilateral Laryngeal Nerve Paralysis and External Arytenoidectomy (PFS 755).* Volume 2.23 (2.55) L. Maximum speeds: Inspiration 0.5 (2) L.P.S., expiration 0.5 (2) L.P.S. Depth Index 2.9 (4) %. Parentheses indicate predicted or normal values.

Deficiencies are present in this method of measurement due to the inertia and momentum of the apparatus and the lack of accuracy, as compared to higher drum speeds and the pneumotachygraph. However, observations on normal subjects have shown maximum air speeds of 2 liters per second and over; observations on patients with appropriate functional impairment have shown air speeds under 2 liters per second.

The one-second, two-second, or three-second volume or proportion of the vital capacity is affected by the progressive slowing of air flow during expiration as well as by the slowing of the maximum speed of expiration. The measuring of the speed of expiration at mid-level or at the tidal air level may or may not be affected by the maximum

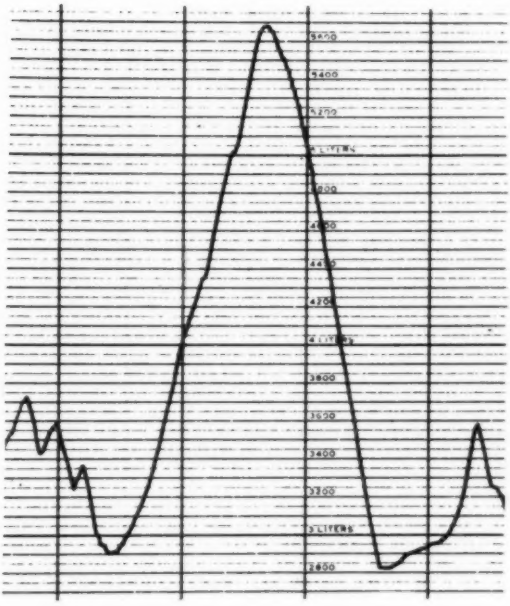


Fig. 3.—*Vital Capacity: Bilateral Recurrent Laryngeal Nerve Paralysis and External Arytenoidectomy* (PFS 666). Volume 2.84 (3.00) L. Maximum speeds: Inspiration 0.3 (2) L.P.S., expiration 0.2 (2) L.P.S. Depth Index 2.9 (4) %. Tracheotomy was required because of respiratory obstruction during an acute upper respiratory infection.

speed of air flow depending upon the level at which the maximum flow stops or the curved portion starts.

The cell block technique developed by Hirsch[2] was extended from the search for tumor cells in cases of suspected bronchogenic carcinoma to evaluation of the type of bronchial inflammation present. The predominance of polymorphonuclear leucocytes is interpreted as an indication of a suppurative bronchitis. The absence or lack of predominance of these cells are interpreted as an indication of a catarrhal type of bronchitis.

A double row edge-punch card (E-Z-Sort Systems, Ltd., Chicago) was developed containing vital information, classification of data from the spirometric examination, diagnoses, special procedures

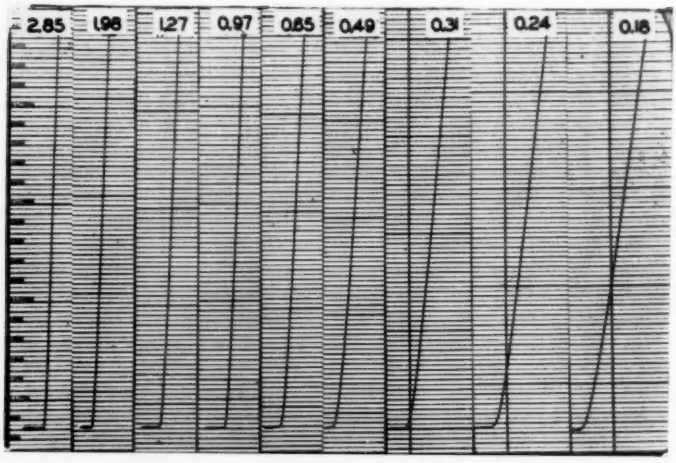


Fig. 4.—Spirometer record with mechanical lung and various orifices.

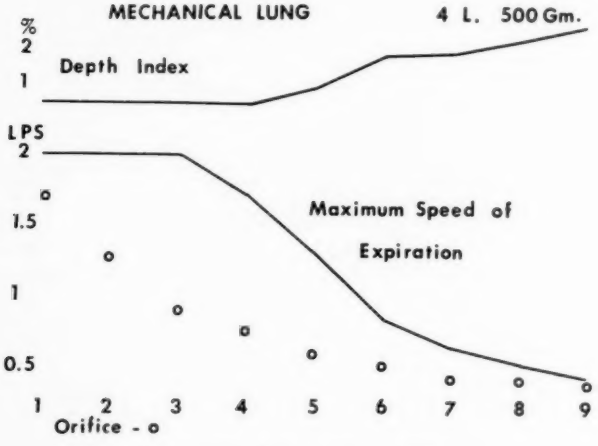


Fig. 5.—Graph of data in Table IV.

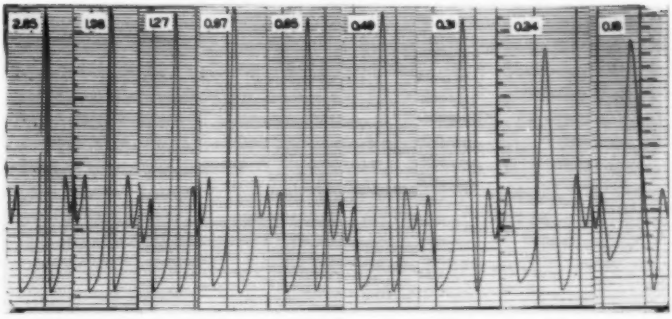


Fig. 6.—Spirometer record with trained subject and various orifices (PFS 116).

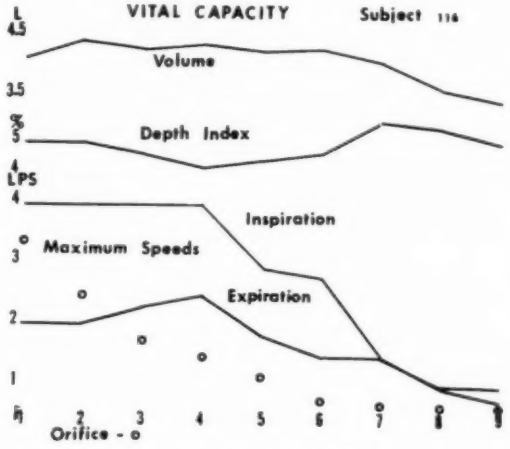


Fig. 7.—Graph of data in Table V.

used and therapeutic responses. The edge-punch card system is thought preferable to other systems because of the numbers involved, simplicity of sorting, and the ability to record data on the individual card thereby avoiding frequent reference to patient records.

A primary diagnosis is made of each patient plus secondary diagnoses as might be pertinent.

TABLE I

PRIMARY DIAGNOSIS AND TYPE OF BRONCHITIS

THE LATTER DETERMINED BY CELL BLOCK HISTOLOGICAL EXAMINATION

Obstructive emphysema		47
Catarrhal	16	
Suppurative	31	
Asthma		33
Catarrhal	13	
Suppurative	20	
Catarrhal bronchitis		19
Suppurative bronchitis		31
Bronchogenic carcinoma		18
Catarrhal	12	
Suppurative	6	

RESULTS

One hundred and seventy-five patients with bronchitis either as a primary or secondary diagnosis are included in this study. The distribution of 148 of the diagnoses are in Table I. The remaining patients had many primary diagnoses.

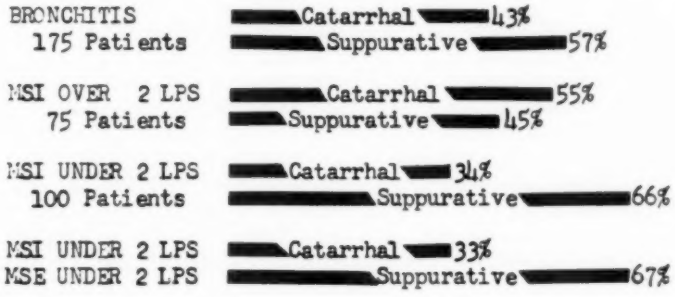
The correlation between the normal and abnormal maximum speeds of inspiration and expiration are displayed in Table II. It is apparent that in the presence of a normal maximum speed of inspiration, the incidence of catarrhal bronchitis is significantly higher than that of suppurative bronchitis. Conversely, in the presence of a slowing of the maximum speed of inspiration, the incidence of suppurative bronchitis is higher than that of catarrhal bronchitis. When there is slowing of both the maximum speeds of inspiration and expiration, the incidence of suppurative bronchitis is still higher.

COMMENT

Studies in bilateral recurrent laryngeal nerve paralysis following external arytenoidectomy (Figs. 2 and 3) reveal a slowing of the maximum speeds of inspiration and expiration but with only a slight increase in the expiratory curve.

TABLE II

MAXIMUM SPEEDS OF INSPIRATION AND EXPIRATION AND TYPE OF BRONCHITIS



A mechanical lung consisting of a weighted rubber bellows is attached to the spirometer and a series of various sized apertures, Table III, are introduced between the bellows and the spirometer. The weight is adjusted to produce a maximum air flow speed of 2 liters per second. The resulting graphs are shown in Figure 4 and the results in Table IV and Figure 5. The smaller orifices produce a slowing of the speed but the expiratory curve is only slightly increased.

TABLE III

ORIFICE SIZES

ORIFICE PLATE IS 0.012 INCHES OR 0.30 MM THICK

Number	1	2	3	4	5	6	7	8	9
Diameter, inches	3/4	5/8	1/2	7/16	23/64	5/16	1/4	7/32	3/16
Area Cm^2	2.85	1.98	1.27	0.97	0.65	0.49	0.31	0.24	0.17

Using the same orifices, vital capacity determinations were done on two trained subjects. The resulting graphs on one subject are shown in Figure 6 and the results in Tables V and VI, and Figures 7 and 8. Again, the slowing of the maximum speeds of inspiration and expiration predominates with only a slight increase in the expiratory curve.

TABLE IV

MAXIMUM SPEED AND DEPTH INDEXES WITH A MECHANICAL LUNG AND VARIOUS ORIFICES

ONLY THE LAST 4.00 LITERS "EXHALED" ARE CONSIDERED. ORIFICES AS IN TABLE III.

Orifice	1	2	3	4	5	6	7	8	9
Maximum speed of expiration LPS	2.0	2.0	2.0	1.7	1.3	0.84	0.63	0.50	0.42
Depth index %	0.5	0.5	0.5	0.6	0.9	1.8	1.9	2.3	2.6

TABLE V

VITAL CAPACITY WITH VARIOUS ORIFICES

TRAINED SUBJECT (PFS 116)

Orifice	1	2	3	4	5	6	7	8	9
Volume liters	4.26	4.42	4.31	4.40	4.24	4.30	4.19	3.63	3.42
Maximum speed of inspiration LPS	4.0	4.0	4.0	4.0	3.0	2.8	1.5	1.0	0.8
Maximum speed of expiration LPS	2.0	2.0	2.3	2.5	1.8	1.5	1.5	1.0	1.0
Depth index %	5.0	5.0	4.6	4.2	4.4	4.7	5.7	5.5	5.0

TABLE VI

VITAL CAPACITY WITH VARIOUS ORIFICES

TRAINED SUBJECT (PFS 1072)

Orifice	1	2	3	4	5	6	7	8	9
Volume liters	3.87	3.77	3.85	3.83	3.77	3.67	3.59	3.62	3.54
Maximum speed of inspiration LPS	4.0	4.0	4.0	4.0	4.0	3.0	2.0	1.4	1.2
Maximum speed of expiration LPS	2.2	2.2	2.9	2.2	2.3	1.8	1.1	1.0	0.8
Depth index %	8.2	11.1	8.8	8.3	7.4	7.9	9.4	6.5	8.4

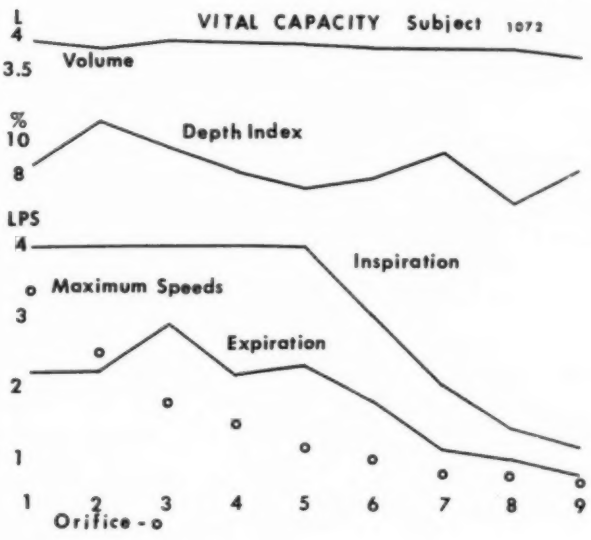


Fig. 8.—Graph of data in Table VI.

From these results it is reasoned that the slowing of the maximum speeds of inspiration and expiration are the result of a reduction in the overall lumen size of the air way. The increased incidence of this abnormality in suppurative bronchitis is strong evidence of an organic type of diffuse bronchial obstruction.

The increase in the expiratory curve, as stated by the depth index, supplies a means of differentiation of two types of obstruction: the organic type causing slowing of the maximum air flow speeds and related to suppurative bronchitis, and the functional type causing the expiratory curve or progressive slowing of air flow during expiration and related to diffuse obstructive pulmonary emphysema.

The recognition of suppurative bronchitis contributing to the pulmonary dysfunction in patients with chronic dyspneic disease supplies a means of selecting patients for intensive therapy designed to liquefy secretion, improve bronchial drainage, and combat bronchial infection. In the authors' experience, this has been of clinical value.

SUMMARY

Using an edge-punch card system, a study of spirometric pulmonary function testing data and bronchoscopic findings has been done.

A correlation has been observed between the maximum speeds of inspiration and expiration of the vital capacity and the type of bronchitis, the latter being determined by cell block histological examination of bronchoscopically collected bronchial secretions.

The correlation consists of a high incidence of suppurative bronchitis in the presence of reduced maximum speed of inspiration and a high incidence of catarrhal bronchitis in the presence of normal maximum speed of inspiration.

This correlation is present in diseases characterized by progressive slowing of air speed as the exhalation proceeds; that is, increased expiratory curve.

The slowing of the maximum air flow speeds without comparable increases in the expiratory curve have been observed in patients with known laryngeal obstruction and demonstrated with a mechanical lung and trained subjects by artificially produced orificial obstruction.

The recognition of a functional disturbance on spirometry as related to suppurative bronchitis has directed therapy to this phase of the disease process and has proved of clinical value.

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REFERENCES

1. Andrews, A. H.: The Functional Examination of Respiration in Obstructive Diseases. Annals of Otology, Rhinology and Laryngology 50:917-928 (Sept.) 1941.

2. Segal, M. S., Herschfus, J. A., and Dulfano, M. J.: Simple Method for Determination of Vital Capacity - Time Relationship. Dis. Chest 22:123-129 (Aug.) 1952.

3. Leuallen, E. C., and Fowler, W. S.: Maximal Midexpiratory Flow. Am. Rev. Tuberc. and Pulm. Dis. 72:783, 1955.

4. Hirsch, E. F.: Organization of the Department of Surgical Pathology. J.A.M.A. 154:881-884, 1954.

XCII

PERMANENT TRACHEOSTOMY IN THE TREATMENT OF PULMONARY INSUFFICIENCY

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Tracheostomy today is one of the most common surgical procedures performed upon both medical and surgical patients. Every medical center of some size now has a so-called "respiratory unit" organized for acute respiratory emergencies, resuscitating patients in respiratory failure and prolonging life in those with respiratory insufficiency. The medical and surgical conditions in which such difficulties arise are constantly expanding. There is an increasing number of patients requiring emergency treatment and specialized after-care in these respiratory units. This involves the use of mechanical respirators usually by way of tracheal cannula with adapter; and so, tracheostomy, cannulization and catheterization of the trachea have become the most essential part of the service of these teams. Today an increasing proportion of emergency cases treated by these respiratory units are patients with chronic pulmonary disease suffering acute attacks of pulmonary insufficiency. For the latter we have recently proposed elective tracheostomy as a part of treatment of progressive pulmonary insufficiency in anticipation of acute episodes. The difficulties and risks of emergency tracheostomy particularly in these patients, have long been recognized.

Our goal here is an elective operation creating an opening into the trachea by means of a mucocutaneous plastic procedure, resulting in a firm stoma that can remain permanent if necessary. Experience of the past few years has convinced us that this goal involves surgical

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and medical problems which still remain to be worked out through co-operation of internist and surgeon.

Our first observations were made over ten years ago on patients with chronic destructive pulmonary disease who had had conventional emergency tracheostomy performed for laryngeal obstruction. By continued use of the metal cannula, the opening was never permitted to close. As lung function deteriorated, these patients became increasingly dependent upon the tracheal opening which permitted frequent suctioning in order to maintain clear airways at all times. We thought then, as most observers still think, that this bronchial toilet was the sole explanation of the beneficial effect of a tracheal opening in critical pulmonary insufficiency. Today we have reason to believe that the matter is more complex than is generally appreciated. During the last two years we have either personally observed or have been in more or less constant consultation upon 24 cases operated upon by several surgeons.

The usual clinical course of these patients is that of progressive decrease of breathing capacity and of recurrent bouts of acute pulmonary insufficiency with critical dyspnea, which may be brought on by a trivial infection (usually bronchial) or overexertion or by mild bouts of circulatory congestion, etc. Any one of these may create an emergency which like an acute respiratory failure in poliomyelitis or trauma or coma, etc., must be overcome by mechanical ventilation and airway clearance in order to forestall death.

Our observations indicate that continued suctioning by way of tracheal opening may prevent the recurrence of acute bouts and perhaps delay the progression of pulmonary insufficiency into pulmonary failure.

The exact indications, contraindications or mode of action of tracheostomy in these patients cannot yet be accurately defined. However there are a few clinical conditions in which we have obtained excellent response and which we consider as indications.

As first indication I should mention the patient with chronic far advanced obstructive emphysema who shows signs of progressive hypoventilation. These patients require urgent vigorous treatment with mechanical ventilators and intrapulmonary suctioning. Both of these are given most efficiently by way of tracheostomy cannula. This is often life saving.

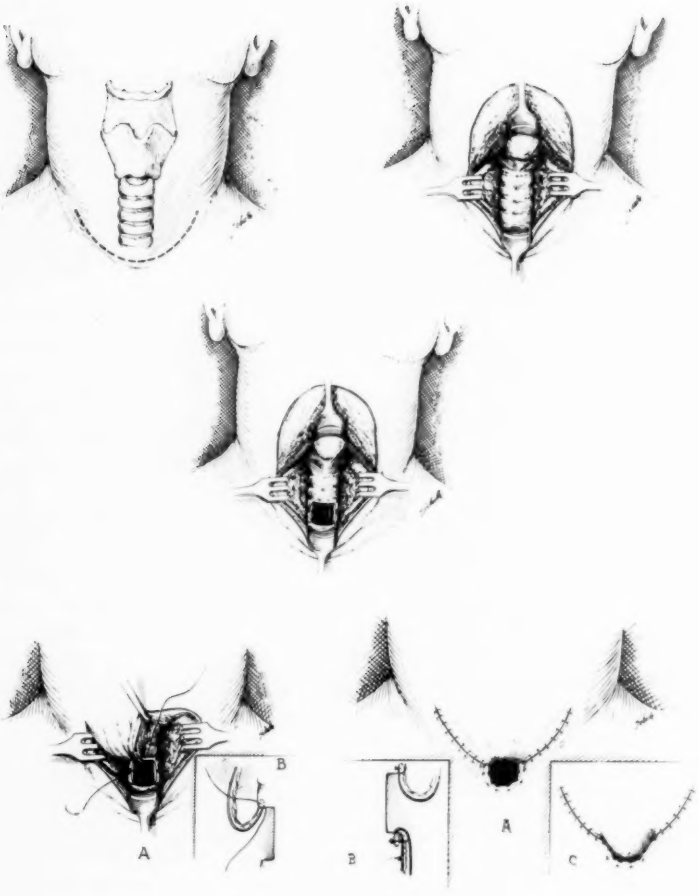
From our experience I should next mention the patients who become pulmonary cripples following pneumonectomy or thoracoplasty. Usually they live comfortably with one lung for some years, until finally they become emphysematous and begin to suffer from low pulmonary reserve. Dyspnea then develops and becomes progressively more severe, associated with persistent exhaustive cough and difficulty in raising sputum. These patients have been enormously benefitted by tracheostomy with suctioning and can go on practically to complete rehabilitation. The tracheostomy has been made permanent here at the patient's request, enabling him to return to work.

A third indication has proved to be a persistent ineffective cough that led to syncope and blackouts in patients with advancing pulmonary insufficiency. In two such patients whom we observed with Dr. Naclerio of New York, a permanent tracheostomy (which he terms "tracheal vent") has prevented further blackouts.

In chronic progressive (obstructive) emphysema, the indications for permanent tracheostomy as well as its mode of action still remain to be defined. Our experience with 18 such patients with far advanced disease showed that nine benefitted, seven progressed relentlessly and two died soon after operation. The benefits were relief of dyspnea, more effective cough with easier expectoration and relief from distressing anxiety associated with spells of smothering brought on by recurrent bouts of ineffective cough. This is true particularly of patients with so-called "vanishing lung" who are apt to be relatively young people with marked dyspnea and dry cough. Following tracheostomy their secretions temporarily increased and often markedly.

We frequently cause increased secretions through suctioning. We have been able to confirm an earlier observation we made, that the more severe the emphysema, the less secretion these patients bring up from the lungs. When secretions are excessive the emphysema is less severe.

Normally the lungs are quite moist organs and have a large fluid circulation. This comes from three sources: 1) serum constantly filters out of alveolar capillaries in amounts which keep the air in the lung fully vapor-saturated; more than a pint of fluid is thus evaporated from the lungs daily; 2) a considerable amount of mucus is secreted normally by the cells lining the bronchial tree; 3) quite recently it has been found that the cells lining the bronchiolo-alveolar junctions constantly secrete a special type of mucolipid substance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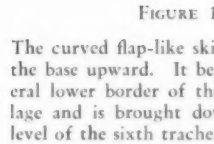


FIGURE 1

The curved flap-like skin incision with the base upward. It begins at the lateral lower border of the cricoid cartilage and is brought downward to the level of the sixth tracheal ring.

FIGURE 2

The skin flap dissected upward, together with the platysma and superficial cervical fascia. The isthmus of the thyroid not shown in this drawing is severed and ligated.

FIGURE 3

Once the trachea is widely exposed, a window-like section, about 2 to 2.5 cm in diameter is resected from the second and third tracheal rings. Before opening the trachea, bleeding areas must be properly ligated and sutured.

FIGURE 4

A. The window-like opening in the trachea with the skin margin of the upper flap being cuffed within the mucosal surface of the trachea. If it is necessary to obtain better mucocutaneous approximation, the lower end of the upper flap may be denuded of its subcutaneous tissue, thus leaving a full thickness flap of skin. *B*. The redundant flap slightly projecting within the tracheal lumen. The tracheal occlusion becomes more marked when the head is flexed.

FIGURE 5

A. The appearance of the stoma when completed. The stitches shown outlining the circular stoma are tied inside of the trachea. *B*. The depth of the skin sutures in the lower part of the tracheal opening. It would have been more illustrative if the artist had reversed this drawing. *C*. The appearance of the tracheal stoma when the head is in the flexed position. This partial occlusion of the trachea allows for normal conversation. When catheter aspiration is necessary, the patient extends the head and the protruding flap moves upward out of the tracheal stoma. Unless infection takes place resulting in the breakdown of the sutured skin within the tracheal lumen, the opening will remain permanent.

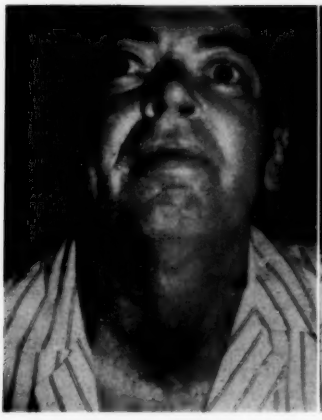


Fig. 6.—Photograph of one of the patients operated on with the head in the extended position showing the appearance of the tracheostomy opening.

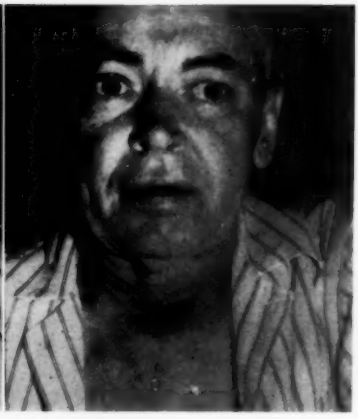


Fig. 7.—The same patient with the head in a slightly flexed position. Note in this photograph the degree of stomal occlusion which makes possible normal conversation.

which serves to suppress surface tension in the airspaces. Most of this moisture keeps flowing upward on the ciliary escalators of the airways in a constant stream. This is accepted as playing an important role in the self-cleansing function of the lungs. In so-called dry emphysema, this fluid stream is diminished. We have found that deep catheter suctioning often starts it up again. The clinical improvement which occurs at the same time, we have assumed to be related to the increased secretion induced by catheterization.

Our observations indicate that in patients with chronic pulmonary disease and copious secretions, there is apt to be severe bronchitis or bronchiectasis with moderate emphysema. Also this combination has resulted more often in pulmonary heart disease than did severe emphysema and moderate bronchitis with scant secretions. Severe bronchitics even with moderate emphysema but with copious expectoration and pulmonary heart disease benefitted little and only temporarily from tracheostomy. Here we were not able, even with frequent suctioning, to get ahead of the copious secretions. We consider suppurative bronchial disease a contraindication to tracheostomy.

We can only speculate regarding the mode of action of tracheostomy with suctioning in these advanced cases of pulmonary insuffi-

ciency. We believe it is more than that of merely relieving secretional obstruction. We also diminish the anatomical as well as the functional dead space and we often stimulate production of secretions. We may be aspirating pockets of trapped air. Perhaps all of these as well as other factors play a part in the final relief of the dyspnea.

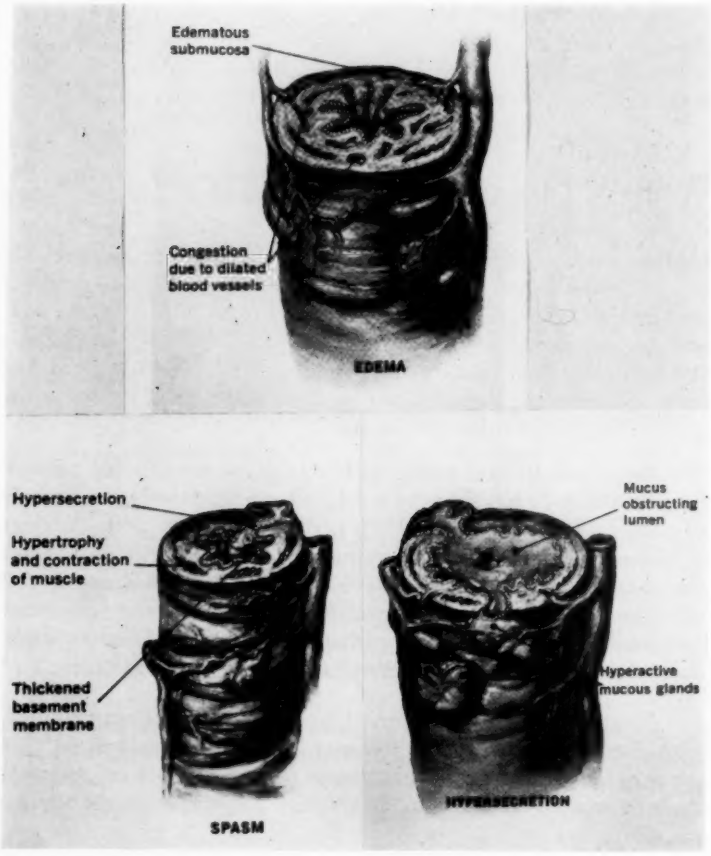
In summary, I should like to stress a few important points. We suggest that the very simplest type of tracheostomy be performed. The technique of this as well as the postoperative management will be described by Dr. Penta. The opening should be made permanent at the start, and later the patient should be given the choice as to whether it be maintained or be closed. A nurse, well trained in postoperative care and suctioning, should be used and such nursing should be available throughout 24 hours of the first week. Finally before operation the patient should be completely informed of the anticipated postoperative discomfort or even distress that accompanies the suctioning, so that full co-operation will be had.

Permanent tracheostomy with suctioning for chronic advanced lung insufficiency has not been employed in a sufficiently large number of patients nor for a long enough period of time to permit dogmatic statements. For the advanced pulmonary cripple, it is chiefly a palliative measure, although the palliation at times may be dramatic. On occasion it has proved to be successful in rehabilitating patients. It frequently fails of its purpose, perhaps because the indications, contraindications and mode of action are not yet accurately defined.

The cases so far treated have been only those critically disabled with far advanced disease. Patients have not yet submitted themselves in the earlier stages of the disease to permit "elective" treatment. Perhaps such earlier intervention can halt progression to the advanced stage.

OPERATIVE TECHNIQUE - POSTOPERATIVE CARE AND GENERAL COMMENTS

Several years ago when Dr. Mayer first proposed a permanent tracheostomy, with catheter suctioning for the palliative treatment of patients suffering from chronic emphysema with pulmonary insufficiency, a number of surgeons attempted to create a tracheal stoma which would remain open without the use of a cannula. It soon became apparent that if the tracheal opening could be so constructed that upon the slightest flexion of the head the tracheal opening would be partially occluded by skin folds or valves, it would allow the patient to carry on normal conversation. In attempting to do this, a number



Figs. 8, 9, and 10.—These three illustrations (shown through the courtesy of the Medical Research Department of Winthrop-Stearns Co.) illustrate the bronchial factors involved in the pathogenesis of chronic obstructive emphysema.

of surgeons devised various forms of ingenious plastic procedures. Many of these procedures proved to be failures, and because the operative time was lengthy, it contributed to the immediate death of the already extremely hypoxic patient.

In discussing this problem with Dr. Mayer, I felt that the desired results could be obtained by a simple mucocutaneous repair of the

tracheal stoma. In the technique of this operation the upper flap is made redundant enough so as to allow infolding into the tracheal opening when the head was slightly flexed. Under local infiltration anesthesia, the skin incision made is a curved flap apron like incision with the base upward. It begins at the lateral border of the cricoid cartilage and brought downward to the level of the sixth tracheal ring. The skin flap is dissected upward together with the platysma and superficial cervical fascia. The isthmus of the thyroid is severed and ligated. Once the trachea is widely exposed, a window like section, about 2 cm in diameter is removed from the second and third tracheal rings. However, this amount of resection is possible only in a large trachea. In smaller tracheas it would be unwise, for obvious reasons. Before opening the trachea, great care must be taken to make sure that all bleeding areas have been properly ligated. As soon as the trachea is opened, the mucosa is anesthetized by spraying directly into the lumen a small amount of 5% cyclaine solution. In some instances where the cough is quite disturbing, a few drops of cyclaine solution is also injected in the lumen of the trachea between its rings before its opening.

In the resection of the anterior and lateral wall of the trachea, enough of the second and third tracheal cartilages should be removed to create an opening, 2 to 2.5 cm in diameter. The important step in the operative procedure is to properly cuff and suture the skin margin to the mucosal surface of the trachea. In this procedure a No. 4 silk suture is used. It is recommended that not too many sutures are used, since this may contribute to infection and breakdown of the tracheal stoma. Having already made a redundant upper flap, there will be some infolding of this flap into the tracheal lumen when the patient slightly flexes the head. This partial occlusion of the tracheal opening will allow the patient to carry on a normal conversation. When catheter aspiration is necessary, the patient extends the head and the protruding flap moves upward out of the tracheal stoma. This may be considered as a sort of check valve mechanism which is controlled by the movements of the head. To insure a better healing of the tracheostomy stoma, a No. 10 short laryngectomy tube is inserted, and worn by the patient for a period of two weeks. The silk sutures are removed on the tenth day. The operative time involved in constructing this permanent tracheostomy varies from 35 to 40 minutes. The entire operation is performed with the patient in the thyroid position (Figs. 1 to 7).

In attempting to describe the technique of this operation before this distinguished group of otolaryngologists, many minute details common to all tracheostomy procedures have been omitted.

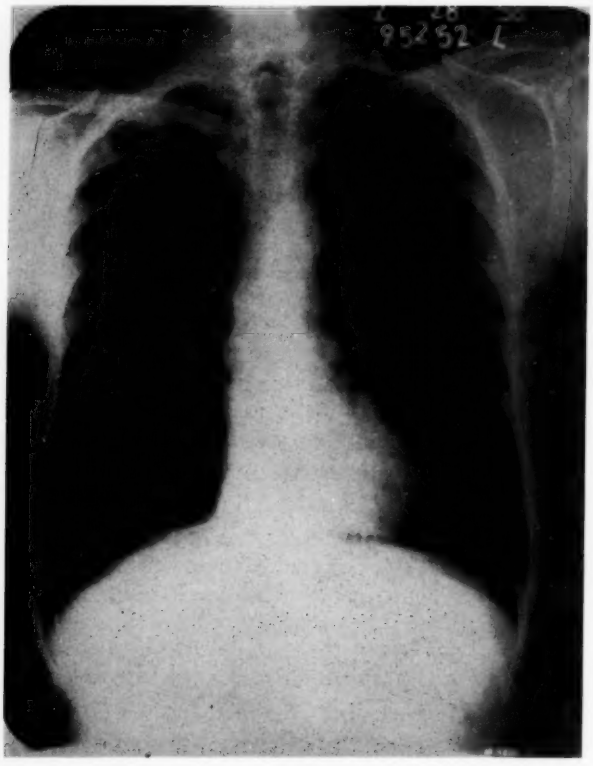


Fig. 11.—Radiograph of the patient in Figure 6, showing a well developed emphysema with marked depression of the diaphragm. He had been critically ill for several months at home before being rushed to the hospital. During the operation the blood exuding from the operative field was "india ink" in color. He survived the operation and following several weeks of hospital care he was discharged in a satisfactory condition.

GENERAL COMMENTS AND POSTOPERATIVE CARE OF TRACHEOSTOMY PATIENTS

Not to repeat, but rather to emphasize some of the important points brought out by Dr. Mayer, I would like to make a few comments on the physiopathologic aspects of chronic emphysema, and to discuss the postoperative management of tracheostomy patients.

The patient suffering from advanced chronic emphysema with pulmonary insufficiency usually manifests symptoms of chronic respir-

atory acidosis due to prolonged carbon dioxide retention. Although the exact cause of chronic emphysema is not generally known, there is good evidence to show that chronic bronchial infection plays a major role in its etiology. Once bronchial obstruction occurs and continues, pulmonary ventilation becomes increasingly hampered. As a result of chronic bronchitis, bronchial narrowing occurs from a hypertrophy of the bronchial muscles due to cough. Eventually there is a permanent thickening of the mucosa from infection which in turn produces mucopurulent secretion. The emphysema resulting from such bronchial constriction and repeated cough may be marked. Then, too, during cough the intra-alveolar pressure is raised as air is forced against a closed glottis, and as the process is repeated many countless times the elasticity of the alveoli becomes diminished. As a result of this derangement of ventilatory function due to bronchial obstruction and loss of alveolar elasticity, air is trapped throughout the pulmonary system creating the so-called anatomical dead space (Figs. 8, 9, 10).

The establishment of a permanent tracheostomy acts as a vent, thereby greatly reducing the closed glottic pressure and the build-up of intra-aveolar pressure. As Dr. Mayer has aptly stated, the distressing cough which is common in this condition is almost completely eliminated. A number of the patients who came under my care developed a transient loss of consciousness due to the violent unproductive cough. The permanent opening in the trachea makes possible constant catheter aspiration of retained obstructing bronchial secretion and reduction of dead space air. It makes possible, if necessary, the continued administration of nebulized bronchodilating drugs and antimicrobial agents directly into the respiratory tract. It is a common observation that the trachea of patients suffering from advanced chronic emphysema is markedly widened and capable of retaining a large amount of mucopurulent secretion. It is not unusual that immediately upon opening of the trachea, there is a sudden gush of purulent material, the removal of which results in immediate relief of the existing dyspnea.

Catheter Aspiration. For the first two weeks these patients require constant aspiration. The nursing care involved is enormous, and unless one is able to cope with the needed hospital and nursing facilities, I would strongly recommend that the operation not be performed. Then, too, the medical management of these patients regarding fluids, electrolyte balance and the judicious use of oxygen proposes a problem of major importance. The type of catheter used in our clinic for aspiration is a polyvinyl plastic catheter, 16 to 18 inches long, and sizes ranging from 12 to 16 French. As soon as the patients start to

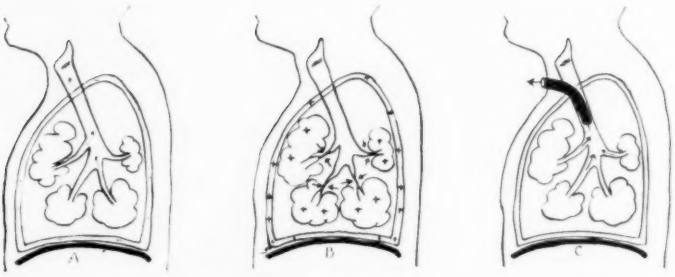


Fig. 12.—*A.* Normal lung at end of expiration or cough. Elasticity of normal lung continues to maintain negative intrapleural pressure which helps to keep the bronchioles patent even at the end of expiration. Outflow of air through opened glottis precludes higher pressures attainable in cough, which is begun when glottis is closed. *B.* Emphysema at end of expiration or cough. Intrapleural pressure now becomes positive rather than negative during expiration. With forced breathing or cough the intra-alveolar pressure becomes increased to such a degree that it compresses the bronchioles. The presence of mucus, bronchiolitis or bronchospasm further predisposes to bronchiolar collapse. This phenomena is referred to as an expiratory "check valve" mechanism. *C.* Emphysema at end of expiration or cough with tracheostomy. Creation of a permanent vent in the cervical trachea greatly reduces closed-glottis pressure and build-up of intra-alveolar pressure. This diminishes or even prevents bronchiolar narrowing or collapse, factors of great importance in airway obstruction.

(This schematic illustration with description of the pneumodynamics of chronic emphysema is taken from a recent article appearing in the New York State Journal of Medicine. It is published here with the permission of the author, Emil A. Naclerio, M.D., of New York City.)

show improvement, they are taught to carry on their own catheter aspirations. It is an interesting observation that these patients soon become skillful in the use of their catheter. They soon learn that by positioning the head they are able to enter either main stem bronchus with ease.

Oxygen Therapy. In the postoperative care of these patients, oxygen must be administered with great care. The injudicious use of oxygen in patients already showing evidence of carbon dioxide intoxication and respiratory acidosis may prove disastrous. The sudden administration of high concentration of oxygen is tolerated poorly and the patient may suddenly develop serious central nervous system irritability and sudden coma.

During the past two years I have had the opportunity of operating on six patients with advanced chronic emphysema complicated

by pulmonary insufficiency. Two of the patients obtained a very satisfactory improvement, and have remained very happy with their tracheostomy opening. In fairness to the rationale of the operation it must be remembered, however, that all of these patients referred to me for this procedure, were already markedly dyspneic, cyanotic, and showed evidence of severe hypoxia. Two of the patients who also had beginning cor pulmonale as a result of pulmonary hypertension brought on by the chronic obstructive emphysema, expired soon after the operation from cardiac failure. The laboratory studies on these patients were limited to blood study only. They were either too ill to be moved or could not tolerate any bedside attempt to carry on any form of ventilatory studies. The result of the blood study in all of these patients revealed an elevated hematocrit, polycythemia, and an increase in the carbon dioxide combining power.

I thoroughly agree with Dr. Mayer when he states that the exact indications for a permanent tracheostomy have not at present been clearly defined. There is no question in my mind that in well selected cases, before there is marked alveolar wall destruction and the symptoms of extreme hypoxia develops, the establishment of a tracheal opening will afford symptomatic relief to many of these patients suffering from one of the most distressing of all pulmonary diseases.

Tracheal fenestration not only helps by reducing the dead air space and the increased intra-alveolar pressure, but it also facilitates the removal of obstructing bronchial secretions. It helps to improve the constant unproductive type of cough which is so exhausting to the already dyspneic patient. It is not unusual for some of these patients during paroxysms of violent spasmodic coughing to develop transitory moments of unconsciousness due to the induced cerebral anoxia. The fenestration, acting like a vent, by-passes the glottic opening, thereby making it easier for the patient to raise sputum on his own power during cough. It must be remembered that there is impairment of diaphragmatic function in many of these patients with advanced emphysema. The diaphragm no longer has the forceful piston-like action to overcome the glottic resistance in the expulsion of secretion. Then, too, with tracheal fenestration the distance which the air has to travel to reach the lungs is shortened (Figs. 11, 12).

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XCIII

LABYRINTHECTOMY

TERENCE CAWTHORNE, F.R.C.S.

LONDON, ENG.

Just over a century ago, Sir William Wilde,[26] writing about the labyrinth in his Text Book of Aural Surgery, said: "It is never the seat of surgical operation and cannot be examined during life." Almost within a generation Kessel[17] was removing the stapes for deafness, and within another generation Jansen[16] successfully drained a suppurating labyrinth. By the end of the nineteenth century, however, operations upon the labyrinth were regarded as potentially dangerous and only justified when suppuration within the labyrinth threatened to spread to the intracranial space.

The next step was to operate upon the non-infected labyrinth for the relief of vertigo, and this was taken by Richard Lake[18] on February 16, 1904. Using the radical mastoid approach all three bony semicircular canals were opened with a burr and as a precaution against sepsis, the wound was swabbed out with Lister's strong mixture, a solution of 1 in 20 carbolic and 1 in 1000 perchloride of mercury. The operation was followed by a brisk reaction, and Lake felt that the shock and irregular movements of the eyes might well have been caused by irritation of the nerve endings in the membranous labyrinth by the strong mixture. The postoperative reaction of Lake's patient was, of course, caused by sudden loss of vestibular function and not by irritation.

I well recall the stormy postoperative course of my earliest labyrinthectomies and how trying this was, not only for the patients but also for those of us looking after them. It usually took three to four weeks before we could get these patients on their feet, and even then they had to walk with a stick for some time. We soon came to realize that in the immediate postoperative period the patient needed to be kept as still as possible; after the first few days graduated exercises, first just in bed, then sitting, and finally walking, hastened recovery.[10]

About the same time as Lake, but quite independently, Sir William Milligan[21] of Manchester also opened the semicircular canals for the relief of vertigo due to non-suppurative ear disease. Both surgeons approached the labyrinth via a radical mastoid which meant that these were open operations.

For some years after this, however, opening the labyrinth was largely reserved for infected ears in which suppuration within the labyrinth had destroyed all function; though occasionally translabyrinthine drainage was tried as a desperate measure in the hope of saving a patient with otogenic meningitis. This was first suggested by West and Scott[25] and I assisted my chief, G. J. Jenkins, in many such operations in the middle 1920's. Apart from such desperate cases it was felt that the labyrinth should never be drained or destroyed in a case of present or past infection so long as there was any sign of life in it, and this view persisted well into the antibiotic age when the possible danger of spreading infection could be overcome, and indeed this view may still be held by some today.

Ballance[2] described the injection into the labyrinth of alcohol for the relief of vertigo, and Mollison (1931) reported two cases of aural vertigo successfully treated in this way. This was a closed operation and gave good results. A variation of this was the injection of alcohol through the tympanic membrane and the footplate of the stapes.[27] This was not without danger to the facial nerve; a danger which existed, though of course to a lesser degree when the alcohol was injected via the lateral canal under direct vision.

In 1943, Kenneth Day used diathermy through an opening in the bony lateral semicircular canal, in order to destroy the membranous labyrinth in Ménière's disease. In this way he hoped to limit

destruction to the vestibular part of the labyrinth, thus preserving hearing.

I started to operate upon patients with Ménière's disease in the autumn of 1938, approaching the bony labyrinth via the mastoid antrum and opening the bony lateral semicircular canal. Then with the help of the Leitz binocular dissecting microscope X10, it was possible to identify the membranous tube inside the bony canal. At first we took samples of perilymph without disturbing the membranous canal if the symptoms were not relieved. They never were, and in one case the patient even developed a Tullio response which only disappeared together with labyrinthine function after the membranous canal had been removed at a second operation.[4] At this time Fitzgerald, Hallpike and I took the opportunity of observing the effect of unilateral destruction of vestibular function on patients. This enabled us to put forward certain views on the influence this had on the response to caloric stimulation of the remaining labyrinth. The results of these observations were published in a series of papers in "Brain" in 1942 on Human Vestibular Function, one of the findings being that Ewald's second Law did not apply to the intact human labyrinth. A summary of this work appeared in the British Medical Bulletin, Volume 12, No. 2, in 1956.

These operations also gave us the opportunity of studying the effect upon the temperature within the lateral semicircular canal of syringing the external auditory meatus with water at 30°C. and at 44°C.[9] This confirmed the views of Dohlman[14] and Schmaltz[23] that thermal changes within the lateral semicircular canal proportionate to the stimulus were brought about by the syringing of the external ear with water both below and above body temperature.

In Ménière's disease removal of the membranous canal was always followed by complete loss of cochlear as well as vestibular function. Even in a few cases, when the membranous tube of the lateral canal was deliberately only torn across, the same thing followed, namely a total loss of labyrinthine function in a labyrinth already severely disordered by the effects of Ménière's disease. It seemed that in Ménière's disease the already disordered labyrinthine end organs were unduly susceptible to any surgical breach of the endolymphatic space; so that total loss of function was the result of such a breach.[5,6] Since then I have had three exceptions to this rule so far as the cochlear

system is concerned. In all three cases pre-operative cochlear function was not seriously impaired in the operated ear; the reason for choosing this operation in such cases being the wish of the patient or his doctor for this operation, even though the possibility of saving the hearing by means of intracranial section of the vestibular nerve had been suggested. In all these three patients, however, there was a deterioration in hearing after the operation, though not a complete loss. In all three, as in all the other 346 patients submitted to this procedure, there was no longer any trace of vestibular function in the operated ear.

Since the autumn of 1956 I have been using a different approach which I mentioned before this Society in 1957, and which has also been described by Schuknecht.[24] In this the approach is as for a stapes mobilization: the annular ligament is incised, the stapes is dislocated and turned aside still attached to the stapedius tendon. Through the open oval window the utricle is found as a flattish sac, the edge of which can just be seen under the facial border of the oval window. This is drawn down by a curved needle and removed with alligator forceps fine enough to pass through the oval window. The identification of the utricle and its removal is not always easy, and I have been interested in the way in which, after removal of the stapes and any excess of perilymph, the utricle retracts upwards underneath the facial margin of the oval window, so that it is almost, and indeed is sometimes entirely, out of sight.

Here again removal of the utricle is usually followed by total loss of both cochlear and vestibular function, though I must report two cases out of 97 in which some vestibular function remained, and in both of these a further operation and removal of more tissue resulted in total loss of remaining vestibular function. I think that it may well be that the utricle had escaped injury at the first operation.

I do not think that as a destructive procedure this is any better than the removal of the semicircular canal, but in view of the general interest in the stapes, it is a golden opportunity to see how the normal stapes, unimpaired by otosclerosis or fibrous adhesions, behaves and how it can be handled with or without damaging it. Moreover it gives the surgeon a remarkable view of the vestibule and its contents which cannot but be of help to him in the various procedures now being undertaken in this region.

By one or other of these approaches I have now destroyed disordered labyrinthine functions in 443 patients suffering from unilateral Ménière's disease, and in only five has there remained any residual function. In three of these five the residual function was in the cochlear division only.

Some years ago, however, I came to realize that this susceptibility to damage by the endolymphatic labyrinth in Ménière's disease was not present when the membranous labyrinth was torn open or removed either accidentally or on purpose in other conditions.[5,6]

For instance, during the course of a fenestration operation for the relief of deafness due to otosclerosis, the membranous canal may be accidentally opened or even torn across. Often in such cases in my experience the hearing has suffered, but I recall at least one in which it was unaffected, and Bateman[3] has recorded a case complete with removed membranous canal in which the hearing was spared.

Then there are the patients with disabling vertigo and an active fistula sign in an old mastoid which I have described under the heading 'Perilabyrinthitis'.[8]

In a few of these removal of the membranous lateral canal was sufficient to end all symptoms, and of course labyrinthine function; but in many symptoms continued or returned, and there was evidence of residual function.

Therefore, in such cases I adopted the principle that all traces of the vestibular end organs should be removed. This includes the ampullae of each semicircular canal, the utricle and the saccule, and is achieved by means of a complete superior and inferior vestibulotomy. Great care has to be taken not to overlook the utricle which always hides underneath the bony bridge containing the facial nerve and which, as I have found out to my cost, can be overlooked. If the utricle is left behind then the patient will remain giddy. I would like to emphasize not only the importance of removing the utricle, but also the ease with which it can be left behind, as it so often lies completely hidden by and plastered against the under surface of the bony bridge containing the facial nerve.

This is in accordance with the findings of Lindsay and Siedentop,[19] who advise removal of all the vestibular end organs, even in

cases of Ménière's disease. My own preference in unilateral Ménière's disease is for one of the simple closed or less radical procedures already described, as they almost always succeed, even though hearing on the operated side is sacrificed.

Now in Ménière's disease it has been, so to speak, the Eldorado of labyrinthine surgery to discover a procedure which will destroy vestibular function but at the same time preserve cochlear function. When there is any cochlear function worth preserving this would be an ideal solution, but often what hearing remains is so distorted that it is a handicap, and many patients declare that they can actually hear better when they have lost it.

Thus in unilateral Ménière's disease, when there is only a remnant of distorted hearing, membranous labyrinthectomy either by the lateral semicircular canal or via the oval window has much to commend it. Should the hearing be good the same operation can still be tried, but I always think that intracranial section of the vestibular nerve, particularly in young and otherwise healthy patients, should be considered, though of course it is a more serious procedure.

Recently the destruction of vestibular function by means of ultrasonic waves has been advocated by Arslan[1] and has aroused considerable interest, as it is claimed that vestibular function is abolished or reduced while hearing is preserved and even tinnitus is diminished.

The outer surface of the bony labyrinth is exposed via the mastoid antrum and the applicator carrying the ultrasonic waves is directed towards that part of the vestibular labyrinth above the facial nerve. From this approach the utricle and the ampulla of at least the posterior canal are sheltered from the ultrasonic waves by the facial nerve, and so they must be spared. Lumsden[20] has said that the ultrasonic method is particularly useful in those cases of Ménière's disease in which the hearing is good and where there is reason to suppose that the disorder is bilateral. It has also been suggested that even if total loss of vestibular function on the affected side is not achieved, the partial loss may be sufficient to relieve symptoms. I have found that complete ablation of vestibular function on the affected side is necessary to ensure freedom from further attacks, and this after all is what the patient expects from surgery. Thus when hearing is poor and when the disease is unilateral, definite destruction of function by

removal of parts of the membranous labyrinth is still the procedure of choice.

My own feeling at present is that further advances in the application and particularly in the focussing of the ultrasonic beam are needed before the method can be generally recommended.

I have found that limited membranous labyrinthectomy as a closed operation in Ménière's disease can be carried out without any danger of infection or of damage to neighboring structures; also that bony labyrinthectomy as an open operation via an old mastoid cavity, even when infected, can also be carried out without any danger of infection spreading to the intracranial space. In Ménière's disease removal of part of the membranous labyrinth is almost always sufficient. In the postsuppurative and postoperative groups removal of all the end organs of the vestibular labyrinth is required.

These operations have been made possible by the use of magnification and chemo- and biotherapy have ensured their safety. They have added to our knowledge of the function of the labyrinth in man and of the appearance and behavior in living man of the structures within it. They have also allowed us to observe the stapes in its natural state unimpeded and unobscured by bony or fibrous adhesions or overgrowth. Finally, the removal and eventual replacement of the stapes is an excellent exercise in the art of handling this fragile little bone.

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REFERENCES

1. Arslan, M.: L'applicazione diretta degli ultrasuoni sui labirinto osseo cura della labirintosi. Minerva Otorhinolaryng. (Torino) 3:141, 1953.

2. Ballance, C. A.: Essays on the Surgery of the Temporal Bone. Macmillan, London, 2:321, 1919.

3. Bateman, G. H.: Case of Successful Restoration of Hearing by Fenestration in Spite of Damage to Membranous External Semicircular Canal. Journ. Laryng. 66:572, 1952.

4. Cawthorne, T.: Treatment of Meniere's Disease. Proc. Roy. Soc. Med. 36: 341, 1943.

5. Cawthorne, T.: The Effect on Hearing in Man of Removal of the Membranous Lateral Semicircular Canal. Acta Otolaryng. Suppl. 78:145, 1949.

6. Cawthorne, T.: Some Observations on the Pathology and Surgical Treatment of Labyrinthine Vertigo of Non-infective Origin. Ann. Roy. Coll. Surg. Eng. 4: 342, 1949.

7. Cawthorne, T.: The Contribution of Surgery to the Problems of Neurology. Brit. Med. Bull. 12:143, 1956.

8. Cawthorne, T.: Adventures with the Stapes. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 66:514, 1957.

9. Cawthorne, T., and Cobb, W.: Temperature Changes in the Perilymph Space in Response to Caloric Stimulation in Man. Acta Otolaryng. 44:580, 1954.

10. Cawthorne, T., and Cooksey, F. S.: Vestibular Injuries. Proc. Roy. Soc. Med. 45:341, 1946.

11. Cawthorne, T., Dix, M. R., Hallpike, C. S., and Hood, J. D.: The Investigation of Vestibular Function. Brit. Med. Bull. 12:131, 1956.

12. Cawthorne, T., Fitzgerald, G., and Hallpike, C. S.: Studies in Human Vestibular Function: II. Observations on the Directional Preponderance of Caloric Nystagmus ("Nystagmusbereitschaft") Resulting from Unilateral Labyrinthectomy. Brain 65:138, 1942.

13. Day, K.: Labyrinth Surgery for Meniere's Disease. Laryngoscope 53:617, 1943.

14. Dohlman, G.: Physikalische und physiologische studien zur theorie des kalorischen nystagmus. Acta Otolaryng. Suppl. 5, 1925.

15. Fitzgerald, G., and Hallpike, C. S.: Studies in Human Vestibular Function. I. Observations on Directional Preponderance ("Nystagmusbereitschaft") of Caloric Nystagmus Resulting from Cerebral Lesions. Brain 65:115, 1942.

16. Jansen, A.: Zur kenntniss der durch labyrintheitering inducirten, tiefen extraduralen abscesse in der hinteren Schädelgrube. Arch. f. Ohrenheilk 35:297, 1893.

17. Kessel, J.: Ueber die durch schreidung des steigbügelmuskels beim Menshen und uber die extraction des steigbügels, resp. der columella bei thieren. Arch. Ohrenheilk 11:199, 1876.

18. Lake, R.: Removal of the Semicircular Canals in a Case of Unilateral Aural Vertigo. Lancet 1:1567, 1904.

19. Lindsay, J. R., and Siedentop, K. H.: Labyrinthine Surgery in the Treatment of Meniere's Disease. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 64:69, 1955.

20. Lumsden, R. B.: Treatment of Meniere's Disease with Ultrasound. Proc. Roy. Soc. Med. 51:617, 1958.

21. Milligan, W.: Meniere's Disease: A Clinical and Experimental Enquiry. Jour. Laryng. 20:105, 1905.

22. Mollison, W.: Two Cases of Vertigo Persisting After Radical Operation Cured by Injection of Alcohol into the Semicircular Canal. Jour. Laryng. 46:191, 1931.

23. Schmaltz, G.: The Physical Phenomena Occurring in the Semicircular Canals during Rotatory and Thermic Stimulation. Proc. Roy. Soc. Med. 25:359, 1932.

24. Schuknecht, H. F.: Ablation Therapy for the Relief of Meniere's Disease. Laryngoscope 66:859, 1956.

25. West, C. E., and Scott, S.: The Operations of Aural Surgery. H. K. Lewis, London, 131, 1909.

26. Wilde, W.: Aural Surgery, Dublin, 371, 1853.

27. Wright, A. J.: Labyrinthine Giddiness: Its Nature and Treatment. Brit. Med. Jour. 1:668, 1938.

XCIV

EFFECT OF MIDDLE EAR MUSCLE ACTION ON CERTAIN PSYCHO-PHYSICAL MEASUREMENTS

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Review of the literature leaves the impression that considerably more is known about the middle ear muscle reflexes in animals than in man. This is due in part to the relative availability of subjects under the desired experimental conditions and to the ease with which cochlear microphonic measurement techniques can be applied to animals.

Perhaps the most surprising and baffling aspect of middle ear muscle physiology is the apparent absence in man of psychological or perceptual changes in the loudness of ordinary every day sounds which presumably are sufficiently high in intensity to elicit the acoustic reflex.

If the middle ear muscles of man act as some sort of loudness decelerator or limiter, they do so in such an imperceptible manner that even a critical introspectionist is unable to experience awareness of such action in our customary ambient noise environment. As a matter of fact, thousands of laboratory measurements by many investigators on human subjects concerned with problems of masking, loudness discrimination and equal loudness level balancing have not disclosed results that appear to be influenced or modified by middle ear muscle contraction. Shall it be concluded that man's intratympanic muscles are less functional than those of animals? Or that the intratympanic muscles do act and modify loudness in an imperceptible manner except possibly under carefully designed experimental conditions?

It appears desirable to present a brief pertinent review of the literature relative to intratympanic muscle action in man before the

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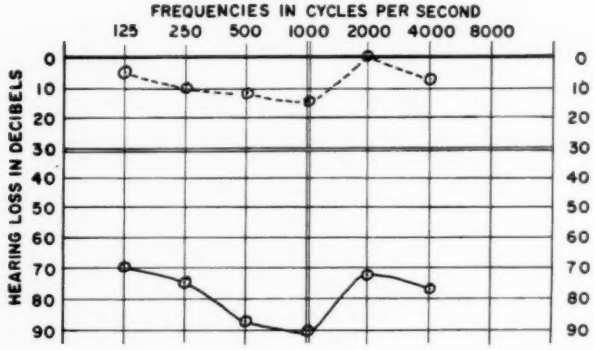

Fig. 1.—o - - - - - - o average threshold sensitivity of better ears of three patients. o————o average hearing levels in better ears at which slight contraction or movement of the stapedius tendon could be observed through perforated tympanic membranes of the opposite ears of these same patients. (After Lindsay, Kobrak and Perlman.)

present experimental data are described. Luescher[7] is credited with the observation that movement of the tendon of the stapedius of man in responses to loud sound stimulation can be viewed through a posterior superior perforation of the tympanic membrane. He also noted that the stapedial reflex can be elicited in the perforated ear by presenting an appropriate acoustic stimulus to the opposite ear. The stapedial reflex has a parallel in the consensual reaction of the pupillary reflex. This consensual reaction of the stapedius was utilized by Lindsay, Kobrak and Perlman[6] in a study of the frequency-energy parameters at which incipient contraction of the stapedial tendon could be observed.

Figure 1 shows the average threshold sensitivity and the average hearing levels required to produce just noticeable movement of the tendons in the opposite perforated ears of three patients. The top dash-dash curve shows the average threshold sensitivity of three approximately normal ears. The bottom solid curve indicates the average hearing levels in the better ears at which slight movement of the stapedius tendon could be noticed through posterior superior perforations in the tympanic membranes of the opposite ears. The intensity differences between the average threshold sensitivity and the incipient

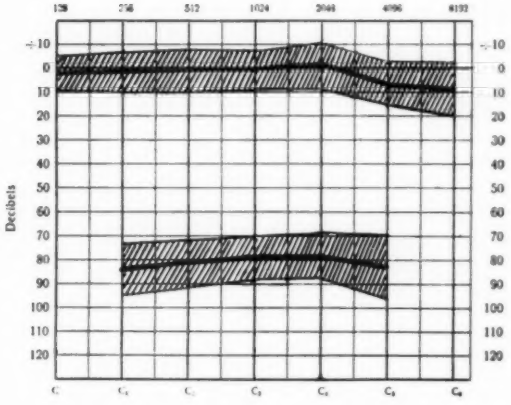

Fig. 2.—Audiogram and threshold curve of the acoustic reflexes of the intratympanic muscles in a normal material. The hatched area around the mean curve represents the dispersion; 95 per cent of the observations fall inside this area.

contraction curves indicate that the stapedial tendon begins to exert observable tension at intensity levels 65 to 75 db above the comparable threshold values.

Metz,[8] one of the pioneers in the measurement of the acoustic impedance of the tympanic membrane in normal and pathologic ears, found that the impedance of the tympanic membrane of a normal ear is altered significantly when an appropriate sound is presented to an opposite normal ear. Figure 2 from Jepson,[3] a student of Metz, summarizes data on 49 subjects with 98 normal ears which indicate the average threshold values and the average minimum hearing levels at which an impedance bridge coupled to one ear is unbalanced when frequencies from 250 to 4000 cps are presented in the opposite ear. The measurements show that tones at the 80 db hearing level over the 250 to 4000 cycle frequency range result in sufficient middle ear muscle contraction to unbalance the impedance bridge used by Jepson.

Figure 3 shows photographs of the relationships between the stimulating sound intensities and the bridge unbalances obtained on

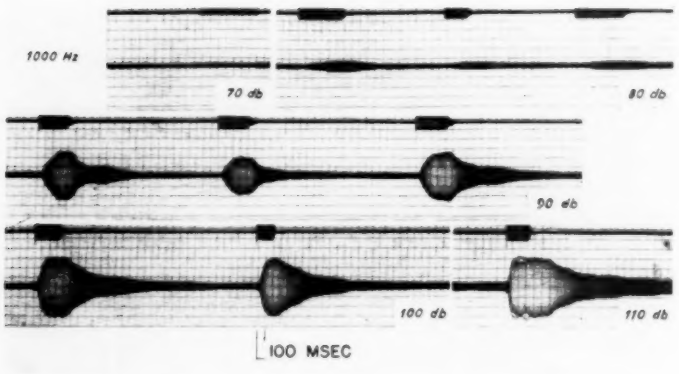

Fig. 3.—Changes in the impedance of the tympanic membrane resulting from contraction of the middle ear muscles while stimulated by a 1000 cps tone at different intensity levels. (From Metz.)

normal ears by Metz.[9] The intensities and durations of the stimulating tones and the synchronized associated impedance imbalances are recorded simultaneously as a pair. The top tracing of each pair indicates the intensity level and duration of the stimulating tone presented to one ear and the lower tracing shows the accompanying change in impedance of the opposite ear. The wider the lower tracing of each pair, the greater the impedance change presumably due to increased tension of the middle ear muscles.

The time interval between the adjacent heavy vertical lines is 100 msec. The top left paired tracings show that the 1000 cps, 70 db sensation level (SL) tone did not result in sufficient intra-tympanic muscle contraction to alter the impedance of the tympanic membrane enough to disturb the balance of the bridge. However, the top right paired tracings at the 80 db SL did result in an imbalance. Additional inspection of the tracings shows that increasing the intensities of the stimulating tones results in progressively greater bridge imbalances up through 110 db SL tone presentations. Careful examination of the tracings reveals that the latency decreases as the intensity of the tone is increased, the rate of contraction increases with increasing

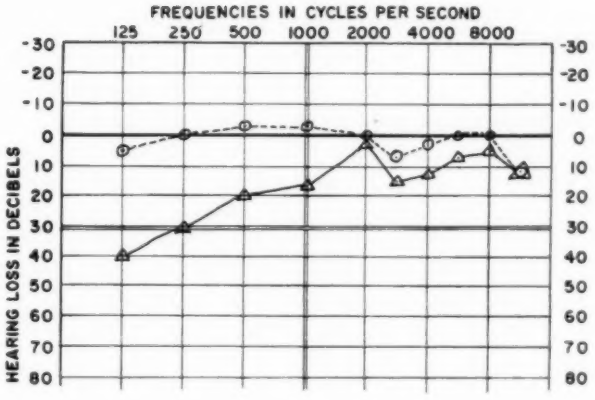

Fig. 4.—o - - - - - - o average threshold sensitivity of both ears with middle ear muscles relaxed. △————△ average sensitivity of both ears during maximum voluntary contraction of middle ear muscles. (One subject, after Smith.)

intensity of the tone, the maximum contraction of the intra-tympanic muscles for a given sound intensity and duration is maintained for an appreciable length of time after cessation of the stimulus and that the relaxation times of the muscles are many times longer than the contraction times at the higher intensity stimulation levels. More detailed analysis of the lowest right paired tracings is instructive. The 1000 cps tone was presented at a 110 db SL for approximately 350 msec. The latency was about 40 msec; the contraction reached a maximum about 40 msec later; the muscles remained in maximum contraction for about 250 msec after the tone was turned off and the relaxation time was prolonged well over a second. It is pertinent to note that Perlman and Case,[10] by recording the action potentials of the contracting stapedius muscle, found the latency of the human stapedius reflex was about 10.5 msec for a loud 1000 cps tone. Consideration of the information inherent in the impedance data presented by Metz[9] must be observed in experiments designed for the study of perceptual phenomena which may be modified by intra-tympanic muscle contraction.

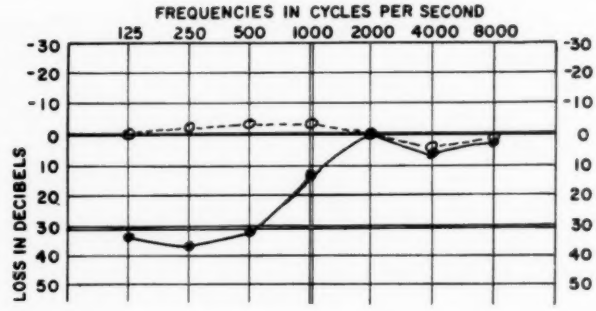

Fig. 5.—o - - - - - - o average hearing level of eight selected ears. ●————● average hearing level of above ears during maximum voluntary contraction of middle ear muscles.

Wever and Lawrence[17] report that Fabricius ab Aquapendente in 1600 stated that he could voluntarily contract his middle ear muscles. Smith,[14] in 1943, presented audiometric threshold measurements on a modern equally gifted contemporary while his intra-tympanic muscles were relaxed and also while they were contracted voluntarily. Figure 4 shows the results of these measurements. Smith states that his subject reported that the 2000 cps tone was increased slightly in loudness while the muscles were contracted, but noted that the audiometric measurements did not seem to confirm this subjective impression.

The writer has approximated Smith's measurements on several subjects. Figure 5 shows comparable data on eight ears of the four subjects out of a group of about twenty who excel in the degree and duration of attenuation of environmental noise when their middle ear muscles are contracted voluntarily. Each of the above subjects was able to maintain voluntary contraction for 20 to 30 seconds before loss of control, at which time renewed effort reestablished the control. Two of the subjects reported a slight increase in loudness of the 2000 cps tone and one a slight increase in loudness of the 4000 cps tone while the muscles were contracted. Individuals highly endowed with this accomplishment report a marked reduction in the loudness of environmental sounds, a change in the tone quality of the sounds

during contraction, and a fluttering noise in their ears, probably arising from middle ear muscle tremors. The above four subjects found that the sound produced by a 20 cps tone in an earphone approximated the sound resulting from intra-tympanic muscle tremors when this tone was presented at the 18 db SL.

Persons who report they are able to contract their middle ear muscles differ markedly in ability to attenuate the loudness of sounds and in the length of time the contraction can be maintained. One of the four subjects reported he could contract the muscles in either or both ears at will. The percentage of persons who have mastered this art is conspicuously low. From the 120 students in the Junior Medical Class at the State University of Iowa, only one or two students are found each year who are able to contract their middle ear muscles voluntarily, but usually in limited degree.

The quantitative values of the attenuation shown in Figures 4 and 5 exceed the approximate 20 db value reported by several animal experimentalists, with the exception of Wiggers,[19] who reported a transmission loss through the middle ear structures as great as 40 db for low frequencies resulting from non-acoustically activated spontaneous middle ear muscle contractions of the guinea pig. It is possible that the transmission loss resulting from spontaneous and voluntary contractions of the intra-tympanic muscles bears little relationship to that due to the acoustic reflex since parts of the neural pathways involved in the different modes of eliciting the reflex must differ in certain respects.

Very few studies on perceptual phenomena resulting from middle ear muscle contraction are reported in the literature. Bekesy and Rosenblith[1] state that a loud high frequency pure tone delivered to one ear will reduce the loudness of a low frequency tone in the opposite ear between five and ten db due to the acoustic reflex. Shapley[11] found that for 30 listeners, all of whom had approximately normal hearing in both ears, the loudness level of a 250 cps tone in one ear was reduced an average of 15 db when a 90 db SL thermal noise was introduced into the opposite ear as measured by means of an equal loudness level balancing technique. However, in a later more carefully controlled study on "Loudness Reduction of Tones Presented to One Ear During the Exposure of the Opposite Ear to Noise" Shapley[12] found that a maximum noise SL of 90 db resulted in a

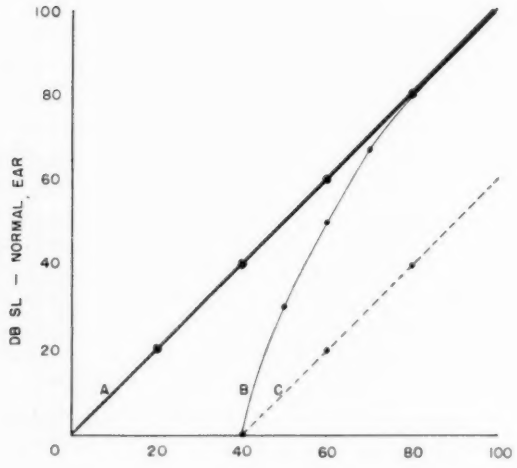

Fig.6—A = alternate ear equal loudness level balances in two normal ears. B = 40 db shift in one ear from masking or cochlear pathology. C = 40 db shift in one ear from tympanic pathology.

maximum mean attenuation of loudness of around 6 db at 250 cps and a maximum mean reverse effect—an increase in loudness—of about 4 db at 3000 cps. Tones at the 60 db hearing level were attenuated essentially as much as tones of the same frequency at 70, 80 and 90 db SL. Subject variability was extremely wide.

Loeb and Riopelle[4] studied the effect of a 2000 cps tone at a 100 db SL in one ear on threshold sensitivity in the opposite ear at 100, 500 and 1000 cps. Threshold shifts were of the order of 3 db. This unexpectedly small shift was responsible for the suggestion that the acoustic reflex may differentially attenuate loud and soft tones. In a follow-up study, Loeb and Riopelle[5] introduced a 2200 cps tone at the 105 db SL in the left ear just before and overlapping the presentation of a 500 cps test tone in the right ear. A comparison tone, identical to the test tone, was introduced a second later. Subsequently, the test tone was varied in intensity and subjects were asked to adjust the intensity of the comparison tones until the test and comparison

tones were equally loud. They found that as the level of the test tone was increased above the 70 db SL, the amount of attenuation, as balanced against the decrease in loudness of the test tone, increased as the level of the test tone was increased. Again, these two investigators concluded that the acoustic reflex provides little or no attenuation for faint sounds but significantly greater attenuation for louder sounds.

The present approach to the psychophysical measurement of attenuation effects of middle ear muscle contraction is a by-product of a comprehensive study of so-called pseudo-recruitment in the normal ear. Steinberg and Gardner[15] pointed out in 1937 that if the threshold of the normal ear be raised or shifted by means of a masking noise, a recruitment-like increase in loudness occurs as the intensities of pure tones are increased above their shifted thresholds. Figure 6 shows the type of nomograph used for presentation of the results of the pure tone equal loudness level balances, obtained on normal, experimental and pathologic ears.

In individuals with two normal ears, a pure tone a given number of db above the threshold of one ear will be equal in loudness to the same tone the same number of db above the threshold of the other ear. This one-to-one linear relationship between the growth of loudness in the separate ears is represented on the graph by the heavy 45 degree line *A*, having its point of origin at the zero hearing level.

The dash-dash diagonal line *C* on the extreme right represents the rate of increase of loudness of a patient in an ear with a unilateral 40 db hearing impairment resulting from tympanic pathology. Alternate ear equal loudness level balance tests reveal that a tone 20 db above the threshold of the pathologic ear (which is at the 60 db hearing level) is equal in loudness to the same tone when heard 20 db above the threshold of the normal ear; and that a tone 40 db above the threshold of the involved ear (which is at the 80 db hearing level) is equal in loudness to the same tone when it is also 40 db above the threshold of the normal ear, and so on. The fact that the straight dash-dash line C is parallel to line A indicates that equal intensity increases above the different threshold sensitivities of the two ears result in equal loudness increases in each ear. Steinberg and Gardner[15] referred to laterally displaced loudness growth curves which parallel line A as being characteristic of constant deafness.

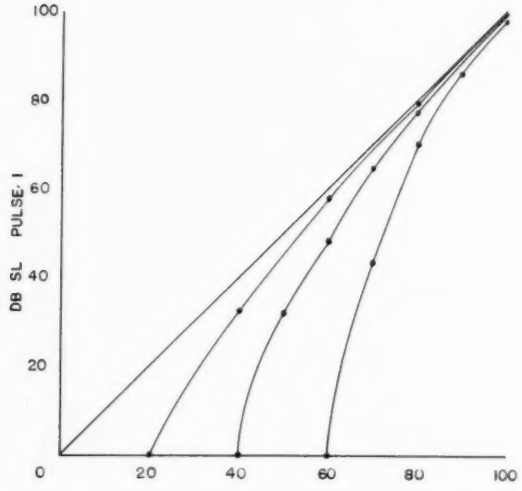

Fig. 7.—Pulse 2 db levels above shifted thresholds. Pulse 1 = 2000 cps. Pulse 2 = 2000 cps plus wide band thermal noise. Both pulses in same ear.

Curve B shows the abnormally rapid growth in loudness in an ear with a unilateral 40 db loss resulting from cochlear pathology. Alternate ear equal loudness level balance tests reveal that the tone 10 db above the threshold of the patient's involved ear (the 50 db hearing level) is judged equally loud as the same tone which is 30 db above the threshold of the normal ear; and that when the tone is 20 db above the threshold of the impaired ear (the 60 db hearing level) it is as loud as the same tone when it is 50 db above the threshold of the patient's normal ear. It is also apparent that a tone which is 40 db above this patient's pathologic threshold is equal in loudness to the same tone when it is 80 db above the threshold of the normal ear. The patient therefore hears the tone at the 80 db hearing level as loud in one ear as the other, despite the 40 db loss in threshold sensitivity in one ear. This abnormally rapid increase in loudness in a pathologic ear is descriptive of the familiar recruitment symptom. Steinberg and Gardner[15] referred to abnormally rapid increase in loudness curves, exemplified by line *B*, as characteristic of variable

deafness. They also obtained the *B* type of loudness growth curve on experimental subjects if the threshold of one of two normal ears were masked or shifted with a thermal masking noise. Steinberg and Gardner[15] did not show the slopes of such curves for masked shifts in excess of 40 db.

The following curves were obtained as the result of measurement on three highly trained and motivated subjects—two graduate students and myself. During the early experimentation, wide band thermal noise was presented in an earphone to shift the threshold of a given test tone a desired number of db. The test tone was then presented simultaneously with the noise at various intensity levels above its masked threshold, which was balanced in loudness against a second tone of the same frequency in quiet. The two pure tones of identical frequency were presented as pulses. Pulse 1, consisting of the pure tone alone, was varied in intensity by means of a 1 db step attenuator by the subject according to the psychophysical method of adjustment and was called the variable pulse. Pulse 2, called the constant pulse, consisted of a pure tone accompanied by noise. The intensity of the masking noise remained constant at a given masking level. The accompanying pure tone was set at the desired levels above its masked threshold while the subject varied the intensity of the variable pulse until the two pure tone pulses were judged equally loud.

The serial order of pulse tone presentation was as follows: variable pulse 1 was one second in duration, separated by a silent interval of 0.2 sec. from the following constant pulse 2, also one second in duration. The time interval between pulse 2 and pulse 1 was 1.5 sec. The serial order of the pulse presentations and the 1.5 sec. silent interval following the pulse containing the stimulating noise was dictated by the relatively slow relaxation or recovery time of the intra-tympanic muscles after intense acoustic stimulation. The 1.5 sec. silent interval also minimized possible auditory fatigue or temporary threshold shift effects. The attenuators which controlled the intensities of the masking noise, and the variable and constant pure tones, were outside the sound proof room where they also could be manipulated and recorded by the experimenter. The setting of the variable attenuator, adjusted by the subject, was varied by means of remote control selsyn motors, so that the subject had no clues to the db values of repeated loudness level balance judgments. The intensity of one of the constant attenuators also could be varied in the same manner by the subject while

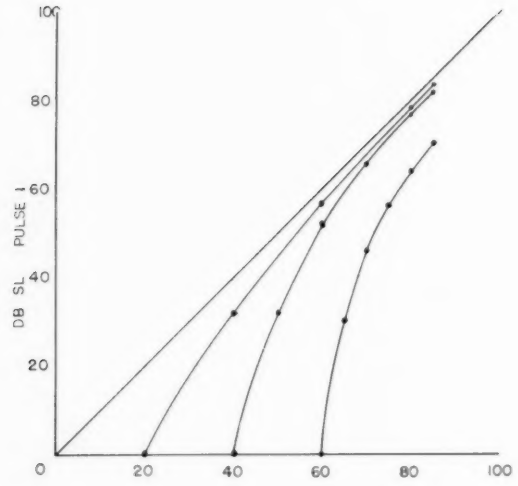

Fig. 8.—Pulse 2 db levels above shifted thresholds. Pulse 1 = 125 cps. Pulse 2 = 125 cps plus wide band thermal noise. Both pulses in same ear.

obtaining his threshold for the constant pulse, after which he was permitted to manipulate only the variable pulse attenuator. The pulsing cycle was repeated automatically by a combination of mechanical and electronic switching.

Figure 7 presents a family of three equal loudness level balance curves which show the rate of increase in loudness in an ear in which the threshold has been raised or shifted 20, 40 and 60 db at 2000 cps by means of a masking noise. The technique for obtaining the 60 db 'growth in loudness' curve on the right will be described in detail. The subject obtained his threshold value for variable pulse 1 by adjusting the setting of the variable 1 db step attenuator until he could just hear the 2000 cps pulse. The threshold in quiet for the 2000 cps constant pulse 2 also was obtained in like manner. The experimenter then increased the gain of the 2000 cps constant pulse to the 60 db SL, after which the wide band thermal noise was increased in intensity until the subject could just hear the 60 db level tone. The intensity

of the constant pure tone was then increased to 10 db above its masked threshold. The subject then varied the intensity of variable pulse 1 until the two pure tones were judged equal in loudness. Three such loudness balances were made by each subject for each intensity setting of constant pulse 2 above its masked threshold. These measurements were repeated two additional times so that each dot on the curves is the average of 27 equal loudness level balances. The 20 and 40 db curves were obtained in similar manner.

Analysis of the 60 db curve indicates that the constant pulse 10 db above its masked threshold was judged equal in loudness to the variable tone 43.5 db above its threshold; and the constant pulse 20 db above its masked threshold was equal in loudness to the variable pulse 70 db above its threshold valve.

The curves of Figure 7 show nothing unusual. They resemble the pathologic recruitment or variable deafness type of curve illustrated on the preceding slide after Steinberg and Gardner.[15] This type of curve also is called an asymptotic curve. An asymptote of a curve is a straight line which the curve approaches continuously in such a way that the distance between the line and the curve approaches zero as they are indefinitely extended. The three curves on Figure 7 are asymptotic curves.

Figure 8 displays a family of curves in which the 60 db masked or shifted curve appears to be a distant relative. These curves were obtained at 125 cps. The masking agent was wide band thermal noise as in the previous illustration. It is apparent that the 60 db masked threshold curve definitely is not an asymptotic curve. The accuracy of this curve has been verified by several repetitions of the measurements. Consideration of factors which might offer an acceptable explanation of the atypical slope recalled the observation that a much louder wide band thermal noise was required to produce a 60 db shift at 125 cps than at 2000 cps. Additional experimentation revealed that an approximate 80 db SL wide band noise was required to produce a 60 db masked shift at 2000 cps but that an approximate 100 db SL wide band noise was required to produce a 60 db masked shift at 125 cps. The following question arose: could the 100 db SL masking noise necessary to produce the 60 db shift at 125 cps have resulted in middle ear muscle contraction which in turn attenuated the loudness of the constant 125 cps pulses?

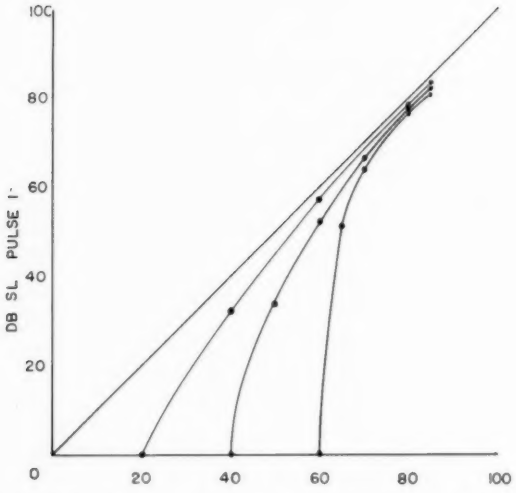

Fig. 9.—Pulse 2 db levels above shifted thresholds. Pulse 1 = 125 cps. Pulse 2 = 125 cps plus 394 cps low pass filtered thermal noise. Both pulses in same ear.

The curves of Figure 9 seems to suggest an affirmative answer to the question. The test parameters of Figures 8 and 9 are identical except for a change in the type of masking noise used. The masking noise used while obtaining the data shown in Figure 9 consisted of filtered thermal noise. The noise was passed through a low pass filter which had an effective sharp cut off above 394 cps. An approximate 78 db SL masking noise was required to shift the threshold of the 125 cps constant pulse the desired 60 db.

Careful examination of the slope of the 60 db shifted curve indicates that this 'growth of loudness' curve approximates the qualifications of an asymptotic curve, which was not the case when the 60 db shift was produced with wide band thermal noise in Figure 8. It is difficult to assume that the rate of increase of loudness above masked thresholds differs with the type of noise used to produce the threshold shifts. This idea, however, remains an open question until answered with additional research.

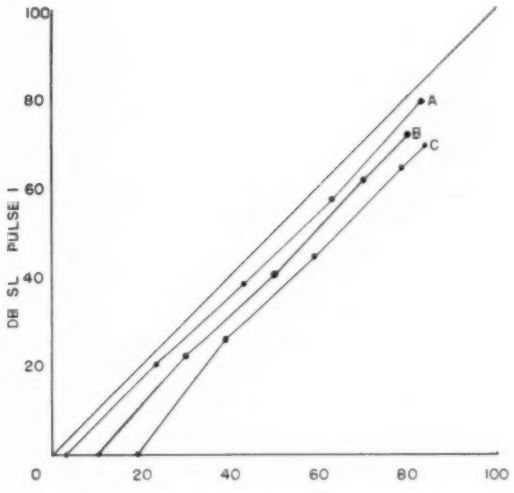

Fig. 10.—Pulse 2 db levels above shifted thresholds. Pulse 1 = 125 cps. Pulse 2 = 125 cps plus 500 cps. Both pulses in same ear. A = 500 cps at 80 db sl. B = 500 cps at 100 db sl. C = 500 cps at 110 db sl.

Figure 10 shows the results of equal loudness level balances at 125 cps when the "noise" consisted of a 500 cps pure tone. It was assumed that the 500 cps tone would produce a minimum of masking of the 125 cps constant pulse and at high intensity levels might elicit the acoustic reflex. Curve *A* shows the slope of the 'growth of loudness" of the constant tone when the 500 cps tone was at the subject's 80 db SL. Curves *B* and *C* show similar data when the 500 cps tone was presented at the 100 db and the 110 db SL's respectively.

It is apparent that these curves are approximately parallel with the 45 degree diagonal and resemble the slopes of curves obtained on subjects with tympanic pathology, so-called constant deafness. It seems reasonable to assume that if the displacement of these curves to the right of the 45 degree diagonal were due to masking, the slopes of the curves would be asymptotic in character.

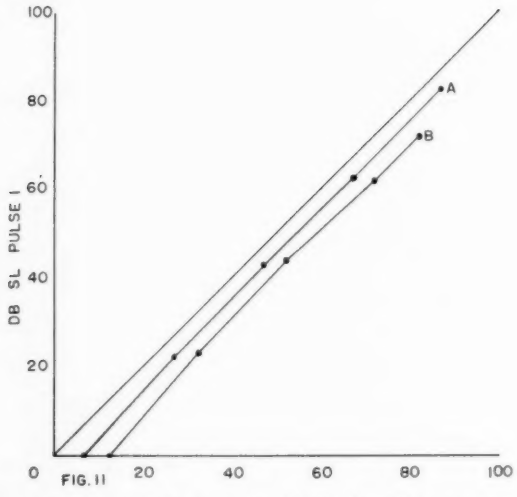

Fig. 11.—Pulse 2 db levels above shifted thresholds. Pulse 1 = 125 cps. Pulse 2 = 125 cps plus 500 cps in opposite ear. Both 125 cps tones in same ear. A = 500 cps at 100d b sl. B = 500 cps at 110 db sl.

Figure 11 shows 'growth of loudness' curves at 125 cps in which the constant pulse consisted of the simultaneous presentation of 125 cps in one ear and 500 cps in the contralateral ear. As shown by the data, the growth of loudness curves are also parallel to the 45 degree diagonal. However, the lateral displacements of the curves are about half as great for comparable SLs of the 500 cps tone as when the 500 cps tones were presented in the same ear and accompanying the constant pulse tone as in the preceding illustration.

COMMENT

The data summarized in Figures 10 and 11 were obtained at 125 cps since animal experimentation and human introspection indicate that the maximum transmission loss resulting from middle ear muscle contraction usually occurs at frequencies below approximately 1000 cps. Inspection of Figures 10 and 11 shows that greater threshold and loudness level shifts occurred when the 500 cps noise was in the

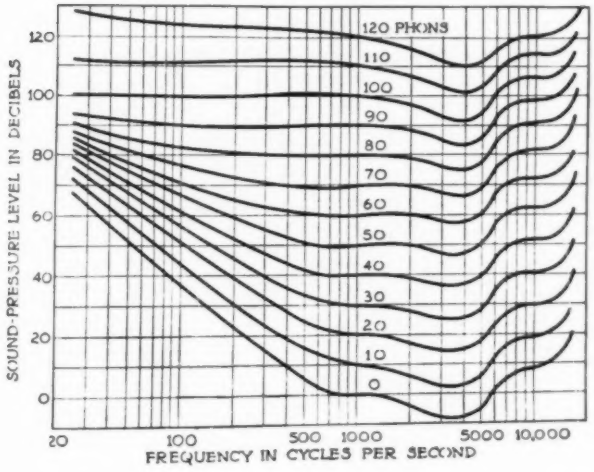

Fig. 12.—Contours of equal loudness. (H. Fletcher and W. A. Munson.)

same ear as constant pulse 2. The greater shifts in Figure 10 do not appear to result from masking, since the equal loudness level balance curves are not asymptotic. It may be that a high intensity level noise presented to one ear results in more effective middle ear muscle contraction in that ear than in the contralateral ear in humans. It was also learned that a high intensity 2000 cps noise had relatively little effect on a constant pulse of 125 cps, even when both were presented in the same ear simultaneously. It may be that a high intensity, high frequency pure tone produces less effective middle ear muscle contraction perceptually than lower frequency tones. Additional parameters await further investigation.

No attempt was made in the present study to differentiate between the relative effectiveness of contraction of either of the two intratympanic muscles. Several investigators report that the stapedius appears to play a much more dominant role than the tensor tympani in altering the transmission efficiency of high intensity sounds through

the middle ear structures of experimental animals. It has been suggested that contraction of the tensor tympani may be involved more intimately with impedance changes of the tympanic membrane because of the attachment of the tensor tendon to the upper part of the manubrium, close to the neck of the malleus.

It is possible that voluntary contraction of the intratympanic muscles produces a more effective contraction of the tensor tympani than of the stapedius. Metz[9] noted that greater middle ear impedance changes resulted from voluntary contraction than from high intensity level stimulation. The writer learned that individuals who can contract their intra-tympanic muscles voluntarily can produce a marked subjective transmission loss by contracting these muscles while exposed to uncomfortably loud low frequency sounds.

Intratympanic muscle tremor noise can be demonstrated in the following manner to most normal hearing individuals who are unable to contract these muscles at will: present an approximate 100 db SL 2000 cps tone in an earphone to a normal ear in a quiet room as a pulse having a duration of at least one second. The pulse should have rapid rise and decay times. The noise made by contraction of the muscles when the tone is turned on, and by relaxation of the muscles when the tone is turned off, usually can be perceived after a few trials.

Animal studies, observations of incipient movement of the stapedial tendon and impedance measurements of the tympanic membrane indicate that middle ear muscle contraction may occur as the result of somewhat lower sound pressure levels than those reported in the present paper. It may be worthy of note that Wersall,[16] as the result of considerable work on rabbits, reported that "the reflex contraction can have a significant effect on sound transmission only after an appreciable tension has been reached." If this observation pertaining to the ear of the rabbit is applicable to the human ear, it is possible that more contraction than that necessary to elicit an incipient stapedial tendon movement or a measurable change in tympanic membrane impedance is required to produce perceptible changes in the loudness of tones.

The data summarized in Figures 10 and 11 show equal transmission losses for both low and high intensity level pure tones since the magnitudes of the threshold shifts and the loudness level balances

formed an approximately straight line parallel with the 45 degree diagonal. It is possible, with certain parameters, that more transmission loss might occur when both the stimulating noise and the pulse tones are at high intensity levels. A combination of a high intensity noise and a high intensity pulse might result in more effective intratympanic muscle contraction than either sound alone.

It appears probable that the slopes of the equal loudness level contours at the higher intensity levels as shown by Fletcher and Munson[20] may be due to middle ear muscle effects. Inspection of Figure 12 shows that the 110 and 120 phon contours, when separated by 10 db steps at 1000 cps, are separated by greater than 10 db intervals at the lower frequency levels.

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REFERENCES

1. Bekesy, G. von, and Rosenblith, W. A.: The Mechanical Properties of the Ear. Handbook of Experimental Psychology, 1951. New York, John Wiley and Sons, Inc., pp. 1075-1115.

2. Galambos, R., and Rupert, A.: Action of the Middle Ear Muscles in the Normal Cat. J. Acoust. Soc. Amer. 31:349-355, 1959.

3. Jepsen, O.: The Threshold of the Reflexes of the Intra-tympanic Muscles in a Normal Material Examined by Means of the Impedance Method. Acta Oto-Laryngol. 39:406-408, 1951.

4. Loeb, M., and Riopelle, A. J.: The Influence of Loud Contralateral Stimulation on the Threshold for Lower Frequency Tones. 1959. Report No. 404, U.S. Army Med. Res. Lab., Fort Knox, Ky.

5. Loeb, M., and Riopelle, A. J.: The Influence of Intense Contralateral Stimulation on the Loudness of a Low Frequency Tone. 1959, Report No. 409, U.S. Army Med. Res. Lab., Fort Knox, Ky.

6. Lindsay, J. R., Kobrak, H. G., and Perlman, H. B.: Relation of the Stapedius Reflex to Hearing Sensation in Man. Arch. Otolaryngol. 23:671-678, 1936.

7. Luescher, E.: Die Funkton des Musculus Stapedius beim Menschen. Zeits. F. Hals-Nasen-Ohrenheilk. 23:105-132, 1929.

8. Metz, O.: The Acoustic Impedance Measured on Normal and Pathological Ears. Acta Oto-Laryngol. Suppl. 63, 254 pp., 1946.

9. Metz, O.: Studies on the Contraction of the Tympanic Muscles as Indicated by Changes in the Impedance of the Ear. Acta Oto-Laryngol. 39:397-405, 1951.

10. Perlman, H. B., and Case, I. J.: Latent Period of the Crossed Stapedius Reflex in Man. Annals of Otology, Rhinology and Laryngology 48:663-675, 1939.

11. Shapley, J. L.: Reduction in the Loudness of a 250 Cycle Tone in One Ear Following the Introduction of a Thermal Noise in the Opposite Ear. Proc. Iowa Acad. Science 61:417-422, 1954.

12. Shapley, J. L.: A Study of Loudness Reduction of Tones Presented to One Ear, During the Exposure of the Opposite Ear to Noise. Ph.D. Thesis, State Univ. of Iowa, 1954.

13. Simmons, F. B.: Middle Ear Muscle Activity at Moderate Sound Levels. Annals of Otology, Rhinology and Laryngology 68:1126-1143, 1959.

14. Smith, H. D.: Audiometric Effects of Voluntary Contraction of the Tensor Tympani Muscle. Arch. Otolaryngol. 38:369-372, 1943.

15. Steinberg, J. C., and Gardner, M. B.: The Dependence of Hearing Impairment on Sound Intensity. J. Acoust. Soc. Amer. 9:11-23, 1937.

16. Wersall, R.: The Tympanic Muscles and Their Reflexes. Acta Oto-Laryngol. Suppl. 139, 1958.

17. Wever, E., and Lawrence, M.: Physiological Acoustics. Princeton: Princeton University Press, pp. 454, 1954.

18. Wever, E. G., and Vernon, J. A.: The Control of Sound Transmission by the Middle Ear Muscles. Annals of Otology, Rhinology and Laryngology 65:5-14, 1956.

19. Wiggers, H. C.: The Functions of the Intra-Aural Muscles. Amer. J. Physiol. 120:771-780, 1937.

20. Fletcher, H., and Munson, W. A.: Loudness, Its Definition, Measurement and Calculation. J. Acoust. Soc. Amer. 5:82-108, 1933.

XCV

THE LURKING LATENT CHOLESTEATOMA

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Recent otologic literature is replete with scholarily scientific dissertations on cholesteatoma etiology. The semantics of "primary" and "secondary" cholesteatomata are still being argued. Histologic, histochemical, roentgenologic and experimental[1-5] forays into the cholesteatoma problem are increasing.

This present discussion of a lurking latent disease is intentionally structured to disturb certain concepts as to what constitutes a safe ear. It is not necessary to belabor the issue that every cholesteatoma is a potentially destructive lesion with many dangerous potentials. Thus, any unrecognized cholesteatoma becomes a lurking threat to its host. We cannot improve on the sage statement made many years ago by Kerrison[6]—"the treatment of cases in which the presence of cholesteatoma can be determined may be summed up in three words—the radical operation."

A reassessment of the term "conservative" is in order. During the past half century many otologists acquired the reputation of conservatism by advising against ill considered mastoid surgery. As a rule they were justified, and the clinical experience of these decades caused the development of some comfortable cliches which were transmitted to students under the neat classifications of "safe" and "unsafe," "non-dangerous" and "dangerous."

Unfortunately, however, many patients who today fall into the safe or non-dangerous category (according to accepted standards)

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are walking around with lurking latent cholesteatomata capable of progressing to unexpected serious complications. Perhaps the conservative attitude in these cases is indeed far from its ideal of saving hearing or life itself. Only the cholesteatoma and its ravages are being conserved. Of course, true conservation must always be our goal, but we must revise our concepts as we acquire new experiences.

I should like to illustrate a number of such instances in which cholesteatomata were lurking behind clinical conditions usually considered safe.

THE DRY CENTRAL PERFORATION WITH NORMAL HEARING AND NORMALLY PNEUMATIZED MASTOID — AN IDEAL MYRINGOPLASTY CANDIDATE

CASE 1. G.W. This man had known of a perforation in his left ear drum for one year. He had no hearing loss. He had no otorrhea. Unsuccessful attempts had been made to close his perforation with paper patches. He was referred to us for routine myringoplasty.

Examination showed a perfectly dry central perforation which approached the postero-superior margin. There was no exudate in the middle ear and the middle ear mucosa appeared to be intact. Hearing was perfectly normal. The mastoid on x-ray examination was well pneumatized and clear. In view of these findings he was considered a good candidate for myringoplasty.

An endomeatal myringoplasty under local anesthesia was started by the usual de-epithelialization of the tympanic membrane. Removal of the perforation margin uncovered a cholesteatoma attached to the postero-medial aspect of the manubrium of the malleus. This cholesteatoma, without an extension to the epitympanum or antrum, was removed, following which a complete exploratory tympanotomy was performed and another small cholesteatoma found in the anterior tympanic compartment. Since no other extensions could be found, myringoplasty was completed with a full thickness graft. Recovery and healing have been uneventful, but the patient will be followed regularly indefinitely.

Myringoplasty is a very satisfying procedure, and one which carries with it a high percentage of successful results, and the enthusiasm in this direction is certainly understandable. Nevertheless it must be pointed out that it is possible for a patient with a completely dry central perforation with normal or near normal hearing to have a tympanic cholesteatoma[7] even in the presence of a normal well pneumatized mastoid.

Obviously a simple myringoplasty done without careful inspection of the entire tympanum would be disastrous in such a case.

Simple closure of such a perforation could lead to dangerous sequellae. With an open perforation the cholesteatoma would at least have some chance for egress and external rather than medial extension. Thus, myringoplasty, without concomitant tympanotomy must be urgently criticized and discouraged.

THE ADULT WITH A DRY CENTRAL PERFORATION, HISTORY OF INFANTILE MASTOIDECTOMY, CONDUCTIVE HYPACUSIS, AND AN INFANTILE SCLEROTIC MASTOID

The patient who has a simple dry perforation with a history of a childhood antrotomy or mastoidectomy will frequently present himself for treatment either because of a desire to have the perforation closed or because of the concomitant hypacusis. In the conductive hypacusis of such cases it is frequently assumed that there has been a dislocation of the incus at the time of the childhood mastoidectomy. In the absence of discharge, pain, vertigo, or any other significant symptom, the possibility of cholesteatoma is usually not seriously considered. That a comfortable security should not be assumed in such instances is illustrated by the following case.

CASE 2. E.B. A 29 year old woman came in because of the primary complaint of a bilateral hearing loss. She gave the history of a right mastoidectomy as an infant and had been aware of a hearing loss, somewhat greater in the right ear. Examination revealed a healed right postauricular incision and a healed dry small right central perforation. Pneumatic compression otoscopy did not produce vertigo or nystagmus. There was a conductive hypacusis bilaterally. Bilateral small sclerotic mastoids were found on x-ray examination. A trial paper patch over the perforation did not improve her hearing. A presumptive diagnosis of ossicular chain discontinuity was made and tympanoplasty advised. She preferred to continue with a hearing aid.

Several months later she returned with an acute suppurative right otitis media accompanied by mastoid tip tenderness. Fortunately she did not respond to conservative therapy with antibiotics, and an exploration of the mastoid was advised. A large cholesteatoma was found, filling the epitympanum and antrum with erosion of perilabyrinthine cells and two small fistulae into the horizontal semicircular canal. A complete radical mastoidectomy was performed with uneventful recovery.

The diagnosis of ossicular discontinuity was correct, but the etiology was not the presumptive dislocation of the incus during infantile mastoidectomy but an unsuspected cholesteatoma. It is also interesting to note that cholesteatomatous semicircular canal fistulization can occur with no noticeable vertigo and no fistula symptoms.

THE ADULT WITH UNILATERAL PROGRESSIVE CONDUCTIVE HYPACUSIS WITH INTACT BUT RETRACTED TYMPANIC MEMBRANE

This is an example of a cholesteatoma lurking behind an intact[8,9] retracted tympanic membrane with the only complaint of a progressive conductive hearing loss.

CASE 3. J.S. This 30 year old male had no history of ear trouble until a temporary loss of hearing in the right ear following an airplane flight. Conservative treatment by an otolaryngologist produced a temporary improvement in his hearing. Several months later there was a return of his loss, following which he was referred to us for examination. Otoscopic findings were bilaterally symmetrical, with intact tympanic membranes quite translucent but rather sharply retracted symmetrically. There was no perforation nor otorrhea in either ear. The right Rinne response was negative and audiometric examination showed a typical right conductive hypacusis. X-ray studies demonstrated a diploic mastoid on the right with some periantral sclerosis and a well pneumatized normal mastoid on the left.

In view of the somewhat progressive history of hypacusis, the x-ray findings and the absence of other findings, a tentative diagnosis of right pseudo-otosclerosis was made and a right exploratory tympanotomy advised.

When this was done, a large monolocular cholesteatoma was found filling the postero-superior aspect of the tympanum and extending from the epitympanum. The drum was replaced and a subsequent mastoidectomy and tympanoplasty performed, at which time the cholesteatoma was removed from the attic. There was an associated antrum granuloma. There was incudomalleolar fusion but the stapes was normal and mobile. A complete radical mastoidectomy was accompanied by Type III tympanoplasty. There was a marked improvement in hearing following surgery.

In retrospect, there were no discernible differences between the two ear drums, the retraction being almost identical. The roentgen difference should have caused some suspicion that this might be pseudo-otosclerosis,[10] and it did. The availability of exploratory tympanotomy as an outgrowth of stapes surgery made the discovery of this cholesteatoma possible long before it had destroyed the stapedial crura, invaded the labyrinth, or transgressed the meninges.

THE PATIENT WHO HAS BOTH OTOSCLEROSIS AND CHOLESTEATOMA

It is possible for a patient to have typical otosclerosis bilaterally, with no history of otorrhea and no tympanic abnormalities. Yet this patient may have a quiescent lurking epitympanic cholesteatoma producing incudo-malleolar fusion in addition to the stapedial vestibular

ankylosis produced by the otosclerosis. The following case report illustrates this diagnostic dilemma and trap.

CASE 4. A.L. This 36 year old man had typical bilateral clinical otosclerosis. He had no otitis history. X-rays showed diploic mastoids with moderate periantral sclerosis.

A right tympanotomy revealed advanced otosclerosis requiring subtotal stapedectomy and prosthetic mobilization. A left tympanotomy revealed advanced otosclerosis and a large monolocular cholesteatoma producing incudomalleolar ankylosis as well. A radical mastoidectomy and semicircular canal fenestration improved his hearing to the 15 decibel level.

It is fortunate that in this patient exploratory tympanotomy for stapes mobilization was advisable and was carried out and the cholesteatoma discovered. The same discovery would have been made had semicircular canal fenestration been undertaken. However, had the advice to the patient been that of the use of a hearing aid, his cholesteatoma would have gone undetected until it had produced invasion phenomena elsewhere in the temporal bone.

THE ADULT WITH DRY QUIESCENT ADHESIVE OTITIS, COMPLAINING ONLY OF CONDUCTIVE HYPACUSIS

A very common otologic problem is that of the patient with a history of infantile otorrhea who presents himself as an adult with a primary complaint of a conductive hypacusis, since he has heard of recent developments in surgery for deafness. He comes in primarily because of his interest in an operation for the relief of his hearing loss. Examination usually reveals the typical findings of complete absence or deformity of the tympanic membrane with a thick scar covering the entire medial tympanic wall. The eustachian tube is usually sealed off by the same scar and there is no discharge in the canal. No cholesteatomatous debris can be seen anywhere and the patient has no history of vertigo, otorrhea or otalgia. His only complaint is deafness. X-rays will usually reveal infantile temporal bones with diffuse sclerosis. This patient, however, may harbor a cholesteatoma in the posterosuperior aspect of the epitympanum or in the antrum as is illustrated by the following case.

CASE 5. E.E. A 50 year old man had a history of ear trouble as a child, but has had no otorrhea, otalgia, or any other active symptoms of ear disease except a hearing loss during the past 35 years. He came in only because of his bilateral conductive loss and was referred as a candidate for stapes surgery by a general practitioner.

Examination revealed symmetrical findings, bilaterally. Both tympanic membranes were hardly recognizable, being represented only by retracted neomembranes with scarring and calcified plaques. There was no otorrhea, the eustachian tubes were patent and allowed inflation of small tympanic pockets. The Rinne was negative on each side, and audiometric examination showed a typical bilateral conductive loss. Both temporal bones on x-ray were infantile in development with generalized sclerosis.

A recommendation was made for right tympanoplasty. Postauricular exploration uncovered a large attic cholesteatoma, accompanied by complete necrosis of malleus, incus, and stapedial crura. A radical mastoidectomy was accompanied by a prosthetic Type III tympanoplasty, with moderate improvement in hearing.

It is interesting to speculate on the outcome of this cholesteatoma. Were it not for recent developments in tympanoplastic surgery, the advice would undoubtedly have been "conservative" and the patient would have been advised to obtain a hearing aid. The cholesteatoma would have gone undetected and the patient subjected innocently to all of the potential sequellae of the disease.

OLD HISTORY OF BELL'S PALSY, DRY ADHESIVE OTITIS, AND MIXED HYPACUSIS

This patient also had a typical adhesive otitis media similar to the one described above, but in addition she had an old Bell's palsy.

CASE 6. S.G. This 64 year old woman had a history of ear infections in childhood, but has had no recent ear complaints with the exception of a constantly progressive hearing loss, which she has known about for at least 40 years. She presented herself for examination in 1957. She complained only of her hearing loss and was interested in surgery for the relief of her deafness. She gave the story of the onset of a left Bell's palsy in 1932 (25 years before examination). This diagnosis of Bell's palsy was made by at least fifteen otolaryngologists throughout the country. No surgery was advised and there was no improvement in the paralysis of the facial nerve. In 1945 a plastic sling operation was performed to improve the appearance of the facial musculature.

Examination revealed a complete left peripheral facial paralysis, a bilateral moderate neural hypacusis with a superimposed advanced left conductive hypacusis. Both tympanic membranes were intact, but markedly retracted and scarred with adherent neomembranes fixed to the medial tympanic walls. Tympanic findings were quite symmetrical. Roentgen examination revealed sclerosis bilaterally, with some attic erosion in both ears, but more pronounced on the left. She was not considered to be a good candidate for left tympanoplasty and no surgery was advised. The patient was told to continue to wear her hearing aid and to come back if she had any further symptoms.

She returned eighteen months later with a new complaint of recent onset of annoying pulsating left tinnitus. Examination revealed a soft tissue swelling in the left posterior canal wall, accompanied by further erosive changes in the left epitym-

panic region on x-ray examination. In view of the new developments a mastoid exploration was advised. An enormous cholesteatoma was uncovered following a postauricular incision. The entire temporal bone was involved, including the petrous apex, with a rather extensive parapetrous[11] extension completely adherent to the temporal lobe dura. There was complete deformation of the middle ear. The incus and malleus were sequestrated. The stapes was rigidly fixed. The round window niche was closed by hyperostotic bone. Extension of the cholesteatoma into the middle fossa was so extensive that it could not be removed in toto by postauricular exposure. It was assumed that this cholesteatoma was probably of primary origin. The facial nerve was virtually absent with the exception of a small amount of sheath in the horizontal portion of the canal and in the genu region. There was virtually no identifiable facial nerve between the geniculate ganglion and the eminentia pyramidalis.

Undoubtedly the diagnosis of Bell's palsy 25 years before we saw her was erroneous. The paralysis was due to an actual atrophy of the facial nerve produced by cholesteatoma pressure. Were it not for the new symptom of annoying pulsating tinnitus in a patient who had no complaint of tinnitus eighteen months previously, an exploration probably would not have been considered advisable or necessary. Certainly the old incorrect diagnosis of Bell's palsy provided a dangerous diagnostic trap for the clinician, as did also the relatively innocuous looking adhesive otitis, which however harbored a lurking cholesteatoma.

BILATERAL ADHESIVE OTITIS, UNILATERAL MIXED HYPACUSIS, AND MILD POSITIONAL VERTIGO

Another problem posed in the commonly occurring adhesive otitis is that of the lurking cholesteatoma producing a labyrinthine fistula, with the only presenting labyrinthine symptom of mild positional vertigo with no nystagmus.

CASE 7. S.H. A 50 year old man has known of a right hearing loss for at least 30 years. Since he had normal hearing in the left ear he completely ignored the right deafness. Two years before our examination he had a severe vertiginous attack treated by his internist as part of a gastro-intestinal disturbance and the vertigo subsided spontaneously. However, several weeks prior to our seeing him he had a recurrence of very mild vertigo, precipitated only by positional change. There was no otorrhea at any time, and there was no otalgia or headache. He was referred to us because of the vertiginous problem.

Examination revealed bilaterally scarred and retracted drums with healed central perforations similarly closed by neomembranes. There was no otorrhea and both tubes were patent on politzerization. The left Rinne response was positive and the right Rinne response was negative. X-rays revealed symmetrical bilaterally sclerotic mastoids. Audiometric examination showed a marked mixed conductive and neural lesion on the right side with normal hearing in the left ear. A cold air caloric test revealed normal latent periods and normal nystagmus durations bilaterally.

In view of the right hearing loss and the vertigo history a right mastoid exploration was advised and carried out. A large multilocular cholesteatoma in-

volved the entire temporal bone with sequestration of ossicles and two large fistulae into the horizontal semicircular canal. The facial nerve was completely uncovered in its vertical portion beginning at the eminentia pyramidalis and extending down almost to the stylo-mastoid foramen. There was extensive cholesteatomatous invasion through the stapedial footplate into the vestibule. A radical exenteration of the total cholesteatotic mass was made, including exploration of the semicircular canal and of the vestibule from which a large cholesteatomatous extension was removed. Obviously no attempt was made to do a tympanoplasty in view of the extensive labyrinthine involvement. Postoperatively his course was quite uneventful except for a mild postoperative vertigo. He has had no further vertigo and his hearing loss has remained unchanged.

There was considerable doubt as to the otologic indication for surgical intervention in this case. However, the decision to open the mastoid was made primarily on the basis of the recurrent vertigo and the rather severe hypacusis of both conductive and neural character on the right side. In spite of normal caloric responses, and in spite of insignificant roentgen findings, the right ear was considered to be potentially the source of the vertigo and the exploration was undertaken.

Here again we find a cholesteatoma in a patient with a healed central perforation with no otorrhea and none of the usual findings which are described as "classic" in cholesteatosis.

Seven years ago the subject of cholesteatosis[12] in children was eloquently discussed before this society by Dr. Philip Meltzer.[13] In reporting 43 cases of children ten years of age or younger, he delineated a number of classic diagnostic features of cholesteatosis in children. He emphasized the fact that hearing in such children may be normal and that the disease may occur in well pneumatized temporal bones. He also stressed the occurrence of cholesteatoma in patients who had anterior or central perforations as was previously shown by Holmes,[14] and he urged early intervention in order to save the hearing.

The next three examples of lurking latent cholesteatomata are in children and each case illustrates a slightly different type of otologic diagnostic trap.

CHILD WITH NORMAL HEARING, NORMAL PNEUMATIZED TEMPORAL BONE, INTACT DRUM, WITH SEVERAL CALCIFIED PLAQUES

CASE 8. C.B. A seven year old child was referred for consultation because of recurrent attacks of bilateral otitis media, accompanying maxillary sinusitis. She palpation of which revealed a fibrous intratympanic mass. There was no history

was examined during a quiescent period, at which time the left drum was normal, the right drum was intact but almost completely replaced by calcified plaques. However, it was mobile and not retracted. There was normal tubal function and hearing was normal bilaterally. Both mastoids were very extensively pneumatized and completely normal.

Four and one-half years later, however, there appeared to be beginning fullness in the posterior one-half of the right drum. There were no changes in hearing, nor were there any roentgen changes. This fullness began to increase gradually. Following an acute respiratory infection purulent otorrhea appeared through a spontaneous posterior perforation. Purulent discharge was accompanied by epithelial debris. Haziness appeared in the right peri-antral region on x-ray. In view of the obvious diagnosis of cholesteatoma exploration was advised. Upon postauricular exploration an enormous cholesteatoma filling the middle ear and attic was exposed. It was accompanied by granulomatosis of the antrum. There was extensive involvement of the long process of the incus and the medial aspect of the tympanic membrane was virtually replaced by the cholesteatoma matrix. In view of these findings, a complete radical exenteration was performed, accompanied by a Type III tympanoplasty with an excellent hearing result.

It is not the intent of this discussion to pursue the conflicting theories of the etiology nor the involved semantics of primary versus secondary cholesteatosis. A number of reports have appeared recently, however, which indicate that these extensive cholesteatomata in children may well be due to metaplastic tympanic phenomena and may not follow the usual conception of epithelial invasion via a marginal or epitympanic perforation. Instances of latent virtually asymptomatic cholesteatoma in children have been reported by Laff[15] and Day.[16]

Obviously, early diagnosis is urgent in these patients in order to intervene in time to safeguard the hearing as well as to prevent serious intracranial complications.

CHILD WITH UNILATERAL CONDUCTIVE HYPACUSIS, INTACT SLIGHTLY BULGING TYMPANIC MEMBRANE

The extensive cholesteatoma in children described above may first make itself apparent only by the slow development of a marked conductive hypacusis as illustrated in the following case.

CASE 9. R.J. This five and one-half year old youngster was examined in a routine school hearing clinic and a report was sent home indicating a left hearing loss. His mother took him to a physician who found a slightly edematous left drum, and he was referred to us for study.

Examination showed an intact but definitely bulging left tympanic membrane,

of otorrhea, or otalgia. The child had not been aware of his hearing loss until the test was made at school. X-ray findings revealed well pneumatized mastoids, bilaterally, with only slight peri-antral sclerosis on the left side. A left exploratory tympanotomy revealed an enormous tympanic and epitympanic cholesteatoma which was subsequently removed completely by means of a postauricular radical mastoidectomy, at which time a multilocular cholesteatoma was found throughout the entire temporal bone. The ossicular chain was entirely necrotic, with no vestiges of incus or stapedial crura. In view of the extensive spread of the cholesteatosis, tympanoplasty was deemed inadvisable at that operation. He has subsequently had several recurrences of cholesteatoma in the mastoid bowl.

This case illustrates the rather rapid ossicular necrosis which can occur with a lurking latent cholesteatoma. In this five and one-half year old youngster there was complete destruction of the ossicular chain, even though the tympanic membrane was intact and even though there was no history of otorrhea at any time.

CHILD WITH ACUTE HEMORRHAGIC MASTOIDITIS

In spite of a good deal of emphasis in otologic writings, pediatricians are still frequently obsessed with the notion that the treatment of acute otitis media is purely medical, and that mastoiditis is a virtually unknown disease. When mastoiditis is encountered, the same emphasis upon antibiotic therapy is urged by many "conservative" pediatricians who decry what they term unnecessary mastoid surgery. Frequently a pediatrician will veto the advice of surgical intervention and will then triumphantly demonstrate a "cured" patient to the somewhat embarrassed otolaryngologist. Many pediatricians are completely unaware of the existence of cholesteatosis and some who realize its existence conceive of it as an extraordinarily rare disease.

Otolaryngologists who do occasional mastoidectomies confined to the postauricular simple operation will not infrequently do a subtotal postauricular mastoidectomy with inadequate exposure of the antrum and completely miss a cholesteatoma. It is possible for an acute mastoiditis to occur superimposed upon a lurking latent cholesteatoma. Such an instance is illustrated in the following case.

CASE 10. C.D. This nine year old youngster had a history of bilateral recurrent otitis and was seen by an otolaryngologist during an acute left otitis. Upon his examination there was considerable mastoid tenderness and the child was referred to us for mastoid surgery. Examination revealed a central perforation through which a small polypoid granuloma and pulsating purulent discharge could be seen. There was marked postauricular tenderness. X-rays revealed a well pneumatized

mastoid with haziness throughout all of the cells, accompanied by significant periantral sclerosis.

At operation an acute hemorrhagic mastoiditis was encountered. There was necrosis of the posterior canal wall and a huge cholesteatoma filled the epitympanum, tympanum and antrum. There was a large extradural abscess with complete necrosis of the tegmen mastoidei. The tegmen was also absent over the tympanum. The malleus was sequestrated and the incus was absent. Hemorrhagic exudate and pus filled all visible mastoid cells. A complete radical mastoidectomy was necessary.

The surgeon who encounters an extensive acute mastoiditis with or without subperiosteal abscess must always be aware of the possibility of an unrecognized unexpected cholesteatoma. No one should do mastoid surgery unless he is prepared by training and experience to continue with the radical operation and tympanoplasty if necessary.

ADULT WITH TWO-WEEK HISTORY OF ACUTE OTITIS

It is not only the child who can present himself with an apparent primary tympanic cholesteatoma with no previous history of otalgia or otorrhea. It is possible for an adult to present himself with an acute otitis media whose primary problem is really an underlying latent cholesteatoma.

CASE 11. T.G. A 25 year old man had an acute right otalgia two weeks before we saw him. He had been treated elsewhere "conservatively" by the use of ear drops and antibiotics and had had no previous history of ear disease. Examination revealed an acutely bulging right tympanic membrane. A myringotomy for this acute otitis media was followed by pulsating purulent discharge with immediate relief of his otalgia. Several days later epithelial debris was found in the canal, along with the pus. X-rays revealed bilaterally diploic mastoids with extensive periantral sclerosis on the right side. There was a marked right conductive hypacusis in spite of the cessation of the otalgia and diminution of otorrhea.

Mastoid exploration revealed a large cholesteatoma filling the antrum, which had destroyed the entire ossicular chain except for the stapes. A radical mastoidectomy and Type III tympanoplasty were followed by uneventful recovery.

This second example of an acute process superimposed upon a latent cholesteatoma should alert us to the fact that in adults as well as children acute otitis media need not be a simple disease.

ADULT WITH TEN-YEAR HISTORY OF RECURRENT AURAL POLYPOSIS, INTERMITTENT OTORRHEA, AND CENTRAL PERFORATION

In most texts today there is a standard teaching that polypi coming from central perforations are usually due to mucous membrane

hypertrophy and that such polyps may be safely removed with an aural polyp snare. In my opinion, this is a most dangerous teaching and one that should never be transmitted to general practitioners or even to general otolaryngologists who are not conversant with otologic surgery. Usually such a polyp will originate in an area of bone necrosis either due to osteitis or osteomyelitis or to an unrecognized cholesteatoma, even though the perforation may be small and central. For an illustration of this problem, let us look at this next, almost fatal, case.

CASE 12. H.S. A 25 year old man had a history of right otorrhea since the age of 15. During these ten years he was seen periodically by otolaryngologists who treated his right ear with local medications and removed polyps on innumerable occasions. He had a number of quiescent periods when the otorrhea ceased and when he had no awareness of ear disease with the exception of a hearing loss. During this time he had about 50 aural polypectomies, always done as office procedures.

When first seen by us, he complained of a stuffy feeling in the right ear, recent weight loss of 20 pounds, generalized malaise, mild otalgia and right temporal headache. Examination revealed a suppurating right middle ear, with granulations obscuring most of the tympanic membrane. X-rays showed a cloudy pneumatized mastoid and audiometric examination showed a 50 decibel conductive hypacusis. The patient was slightly lethargic and moderately tender over the mastoid tip and antrum.

In view of the rather ominous findings, an immediate mastodectomy was advised and performed. There was a marked destruction of all cells with granulomatous degeneration of the entire mucosa of the mastoid and middle ear. There was a large central perforation. Thin seropus filled most of the antrum and peri-antral cellular area. There was a small cholesteatoma confined to the round window niche with extension into the region of the hypotympanum and into the obturator foramen and anterior stapedial crus. There were three distinct erosive fistulae in the horizontal semicircular canal, and one large fistula in the superior vertical canal. There was a large extradural middle fossa abscess with a granular middle fossa dura.

Following complete radical mastoidectomy, his course continued downhill and he developed a complete contralateral hemiplegia within 48 hours. Neurosurgical exploration through craniotomy revealed a temporal lobe abscess with 160 cc foul pus. Subsequently, other abscesses in the occipital and frontal lobes were drained. Following a very stormy course, he finally began to make a slow recovery. He still has a residual partial left hemiplegia, but his craniotomy wounds have healed and his mastoid wound is completely epithelized.

Here we are dealing with a patient with a central perforation and recurrent aural polyposis. Repeated polypectomies gave him periods of reasonable freedom from otorrhea and from other ear symptoms except conductive hypacusis. During this entire ten year period, the round window cholesteatoma was undoubtedly the cause of the polyposis and the granulomatous mastoiditis. The unrecognized choles-

teatoma was undoubtedly the cause of meningeal extension and the development of the temporal lobe abscess.

So we see an almost fatal sequel in a patient who had what has been described repeatedly as a "safe" ear, and for which he was treated by conventionally accepted methods of treatment, namely aural polypectomies. Indeed such "conservatism" almost cost this patient his life.

TYMPANOPLASTY FOR GRANULOMATOUS NON-CHOLESTEATOMATOUS MASTOIDITIS WITH OSSICULAR NECROSIS — IATROGENIC CHOLESTEATOMA

Last but not least we must consider the case of the iatrogenic cholesteatoma, the one created by the otologic surgeon himself. We are beginning to see this, not only in tympanoplastic surgery but even in stapes surgery. As an example of this, we will look at the following case.

CASE 13. S.B. This 41 year old man had a right dry adhesive otitis with conductive hypacusis. He was considered to be a good candidate for reconstructive surgery and a radical mastoidectomy and Type III tympanoplasty were performed. At the time of surgery chronic granulomatous (non-cholesteatomatous) mastoiditis and otitis were encountered along with ossicular necrosis. A split thickness graft was used for the tympanoplasty. Postoperative healing was uneventful but the graft became adherent to the promontory. Accordingly, 14 months later a revision was performed at which time a cholesteatoma was found in the retrofacial region. Following removal of this "iatrogenic" cholesteatoma, a full thickness skin graft was used to recreate a Type III tympanoplasty with an uneventful recovery and a gain in hearing.

This case illustrates what has been reported quite frequently during recent years by skilled tympanoplastic surgeons, both in this country and abroad.[17]

We must not approach the subject of tympanoplasty lightly. Not only will we be seeing recurrent cholesteatoma due to inadequately removed cholesteatosis prior to the tympanoplastic procedure, but we will see, as in the above case, cholesteatoma developing under a graft in a patient who never had cholesteatoma in the first place. This skin graft itself became the cause of the subsequent postoperative cholesteatoma, truly an example of iatrogenic otologic disease and quite unsuspected. Thus, lurking latent cholesteatoma may be implanted by the otologist himself, and he may be unaware of it.

SUMMARY AND CONCLUSIONS

Cholesteatoma is a serious disease of the temporal bone and always demands radical removal.

Cholesteatoma may lurk behind central perforations, behind dry ears, and in many other conditions commonly described as safe. A reassessment of otologic thinking regarding cholesteatoma and conservatism is overdue.

A number of instances are cited in which conventional otologic standards would have pointed to conservative management and where cholesteatomas would not have been seriously considered. In each of these a cholesteatoma was present with serious consequences either potential or already in existence. The possibility of ignoring such lurking latent cholesteatomata should constantly haunt the conscience of the otologic surgeon, for his well meant "conservatism" may be anything but truly conservative since he will conserve neither the patient's security nor the patient's hearing. Obviously this does *not* call for a sudden increase in radical temporal bone surgery with abandon. On the contrary, it calls for far more attention to detail and far more meticulous consideration of all possibilities when examining the patient with otologic abnormalities. Cholesteatoma is not a rare disease. It is one of the most common diseases of the temporal bone and its presence must always be considered until ruled out by appropriate measures.

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REFERENCES

1. Friedmann, I.: The Comparative Pathology of Otitis Media, Experimental and Human. II. The Histopathology of Experimental Otitis of the Guinea Pig with Particular Reference to Experimental Cholesteatoma. J. Laryng. Otol., Lond., 69:9: 588-601 (Sept.) 1955.

2. Schroer, R.: Particularities in Experimental Production of Cholesteatoma. HNO, Berl., 6:4:116-7,12 (Apr.) 1957.

3. Beikert, P.: Hernia Like Formation and Development of Cholesteatoma. Zschr. Laryng. 36:3:154-64 (Mar.) 1957.

4. Fernandez, C., Lindsay, H. R., Moskowitz, M.: Some Observations on the Pathogenesis of Middle Ear Cholesteatoma. A.M.A. Arch. Otolar. 69:5:537-46 (May) 1959.

5. Ruedi, L.: Cholesteatoma Formation in the Middle Ear in Animal Experiments. Acta Oto-laryngologica, Stockh., 50:3-4:233-40 (May-Aug.) 1959.

6. Kerrison, P. D.: Diseases of the Ear. J. B. Lippincott Co., Phila., p. 215, 1913.

7. Temesrekasi, D.: Zentrale Perforation und Cholesteatom. Pract.-oto-rhino. 17:271-281, 1955.

8. Keru, G.: Zur Entwicklung des Cholesteatoms Hinter Intaktem Trommelfell. Schwz. Medizinische Wochenschrift 32:783-784, 1958.

9. Anderson, H.: Cholesteatoma and the Sealed Tympanum. J. Lar. Otol. Long. 70:8 (Aug.) 1956

10. Goodhill, V.: Pseudo-Otosclerosis. Trans. Amer. Otol. Rhin. and Laryng. Soc., in press.

11. Preçechtel, A.: Epidural Parapetrous Cholesteatomatous "Layer": A Special Pathological and Clinical Entity. Acta-Otolaryngologica 46:340-351, 1956.

12. Walsh, T. E., Covell, W. P., Ogura, J. H.: The Effect of Cholesteatosis on Bone. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:1100-1113, 1951.

13. Meltzer, P.: Unrecognized Cholesteatosis in Children. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:1174-1185, 1953.

14. Holmes, E. M.: A Review of 303 Cases of Cholesteatoma. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:135, 1938.

15. Laff, H. I.: Two Cases of Primary Latent Cholesteatoma Diagnosed and Treated by Tympanoplasty. Laryng. 69:6:682-692, 1959.

16. Day, K. M.: A Primary Cholesteatoma of the Middle Ear. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 64:161-3, 1955.

17. Beikert, P.: Das Lappencholeastom - Eine Spaet Komplikation Nach Tympanoplastik. Zeit. für Laryngologie, Rhinologie, Otologie 37:567-573, 1958.

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XCVI

SOME CLINICAL ASPECTS OF APHASIA

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It must be admitted at the outset that aphasia is a complex disorder. However, on many occasions the true complexity is compounded by the use of long and complicated terminology and by failure to define terms. Further complications occur when dogmatically rigid viewpoints are upheld too vigorously. I hope to be able to present a simple, flexible, and accurate evaluation of some of the clinical aspects of aphasia.

Aphasia may be defined as a primary disorder of the reception, manipulation, and expression of language. The word "primary" is used to exclude language disturbances which are secondary to special perceptual defects such as deafness, special motor loss of the speech musculature, general intellectual deficits as exemplified by dementias, and emotional disorders such as neuroses and psychoses. The phrase "reception, manipulation, and expression" is used specifically to imply that in almost all aphasic patients there is involvement of more than one aspect of language. The terms "reception" and "expression" are generally well recognized. The term "manipulation" of language is used to designate the conceptual language processes going on between reception and expression. These conceptual language processes have

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been variously designated as inner speech, inner language, symbolic formulation and so forth.

Language may be defined as a system of communication of ideas and feelings by the use of empiric symbols known as words. Some of the special forms of language are sign language, braille, mathematics and chemical formulas. In most aphasic patients concern is concentrated on the usual forms of language.

Language may be considered to consist of four major modalities: listening, speaking, reading, and writing. In most aphasic patients more than one language modality is involved.

LATERALITY OF LESION AND HANDEDNESS

It was recognized early that aphasia resulted from lesions limited to one hemisphere, usually the left. Subsequently possible interrelationship between handedness and the laterality of the lesion producing aphasia has been studied. There has been a remarkable divergence of views on this apparently simple point.

Nielsen,[1] an exponent of the viewpoint of detailed localization of various cerebral functions, stated, "In general, right-handed persons are left-brained and left-handed persons are right-brained; the proportion of instances in which brainedness and handedness are ipsilateral is small, perhaps 5%."

Wepman[2] expressed a contrary view. "Language deficits following damage to the right hemisphere either are of the nature of dysarthrias or, if aphasic-like last only a short time and are overcome spontaneously. Permanent aphasia . . . occurs only after injury to the left cerebral hemisphere, regardless of cortical dominance."

These two statements are illustrative of the wide diversity of opinion concerning the laterality of lesions producing aphasia. There are, of course, various opinions lying between these two extremes. In a reported study of 100 aphasic patients,[3] adequate evidence of a unilateral lesion which could be accurately lateralized was found in 93 patients. In 93.5 per cent of the group the lesion was in the left hemisphere; among the right-handed patients the lesion was in the left

hemisphere in 96 per cent and in the right hemisphere in 4 per cent, while among the left-handed and ambidextrous patients the lesion was in the left hemisphere in only 73 per cent and in the right hemisphere in 27 per cent.

In six patients, aphasia was due to a lesion of the right hemisphere and the aphasic phenomena seen in these patients were no different from those seen in patients suffering from aphasia due to lesions of the left hemisphere. In one patient with a lesion in the right hemisphere which produced moderately severe aphasia, the onset had occurred eight months before his examination.

Penfield and Roberts,[4] on the basis of tests with sodium amytal and evidence from cortical excisions for epilepsy, estimated that less than 1 per cent of the right-handed population have speech represented in the right hemisphere while less than 10 per cent of the left-handed persons have some, and probably all, speech represented in the right hemisphere.

Further work with larger series of patients will be required to establish the percentages more exactly, but it seems safe to assume that among the right-handed population, aphasia in most patients in whom it develops is the result of a lesion of the left hemisphere. Among left-handed and ambidextrous patients, aphasia usually will be the result of a lesion in the left hemisphere and in contrast to right-handed patients, only a small although a definite percentage of left-handed and ambidextrous patients will have aphasia as a consequence of a lesion of the right hemisphere.

LOCALIZATION OF LESION

The localization within the hemisphere of lesions producing aphasia has similarly been the subject of controversy.

Nielsen[1] defined a cerebral center as "an anatomically delimited zone which is essential to a certain function." With such a definition in mind he cited evidence for the existence of centers for specific language skills including visual language receptive skills, auditory language receptive skills, language formulation, spoken language expression, written language expression, and visual object recognition.

In some contrast to this, Wepman[2] accepted the view that "aphasic language disorders are considered as affecting all the language modalities and cannot be limited to specific language skills."

An intermediate view has been proposed more recently by Penfield and Roberts.[4] Their evidence is based on obtaining aphasic responses while stimulating the cerebral cortex electrically and on studying the language of patients treated by cortical excision for focal epilepsy. They concluded that three cortical areas are of particular importance in language functions. Of greatest importance is a posterior area comprising the posterior temporal and adjacent parietal lobe. The area of Broca is the second region in importance. Of still lesser importance is the supplementary motor area on the medial aspect of the hemisphere. They further observed that lesions near the junction of the parieto-occipital lobes (the posterior portion of their posterior area) may produce dysphasia with most pronounced difficulty in the visual sphere.

A study of 94 patients with aphasia in whom an accurate localization could be made on clinical evidence was reported in 1957.[3] In only three patients did the lesion producing the aphasia seem limited to the frontal lobe or adjacent temporal tip. In the remainder of the patients, lesions occurred more posteriorly and involved the midportion and posterior portion of the temporal lobe and the adjacent parietal or occipital regions. In 13 patients an aphasic defect of reading was considerably greater than other defects. In 10 of the 13 the lesion responsible for the aphasia was localized posteriorly and involved the posterior temporal anterior occipital region. Patients with predominant defects of reading were most frequently associated with homonymous hemianopsia suggesting a relatively posterior location of the lesion. Patients with severe defects of all language modalities (global aphasia) had the greatest frequency of hemiplegia and hemianesthesia suggesting a generally more massive lesion.

Although there are many details of localization which have not been touched on in this discussion, these are a few general principles which stand out. Aphasia may occur as the result of a lesion strictly limited to the frontal lobe, but such a location is statistically uncommon. By far the commonest location for a lesion producing aphasia is in the midportion and the posterior portion of the temporal lobe and adjacent to the parietal lobe. A lesion limited to the more pos-

terior part of this general region tends to be associated with aphasia in which defects of reading are more prominent than the other aphasic defects.

DESCRIPTION OF APHASIC DEFECTS

Over the years a wide variety of systems of classification of aphasia have developed, often as a result of different points of view and with varying usefulness. Some classifications were designed for purposes of anatomical localization, others for purposes of therapy. For most physicians with not too deep an interest in aphasia it is perhaps satisfactory to bypass systems of classification. It is important, however, to examine the patient for the presence of specific aphasic defects. In so doing, tests should include all four major language modalities: listening, speaking, reading, and writing.

Because it is most obvious, speaking is generally tested first. Abilities of speaking occur in a continuum of increasing difficulty, but for purposes of demonstrating severity of aphasic defects it is convenient to divide the continuum into separate groupings.

The lowest level of speaking might be categorized as *automatic speech.* It includes phonatory noises, exclamations, swearing, and stereotyped speech remnants. One patient with such a stereotyped remnant responded to everything with the words "baby shoes." Automatic speech of this character may convey emotional feeling but does little in conveying a variety of specific conceptual thoughts.

The next level of speaking can be characterized as *socially reactive speech.* It includes the ability to use "yes" and "no" meaningfully and to comment by parenthetical asides. One patient, after repeated failures during a therapy session, murmured parenthetically, "I hope I talk soon." The ability to react verbally to a situation of considerable personal meaning occurs at this level, as exemplified by a hospitalized patient, unable to speak volitionally, who, when presented with a dish of pudding, exclaimed, "No, I don't want that—I don't like it!" Conversing in generalities (often vaguely) about familiar daily events is also included at this level. When such socially reactive speech remains, a patient may not be recognized as aphasic by the casual observer.

The ability to utter the desired specific word is the third level of speaking. This might be designated as the level of *specific speech.* Included in this level is the ability to name objects accurately, to use the proper verb to describe an action, and to use the correct modifying adjectives or adverbs. Defects at this level are illustrated by the patient who was asked the name of an object (razor) and responded with "Well, to shave with." When asked what you do with a razor he said, "Well, on your face." Defects at this level are readily tested and are readily recognized as aphasic.

The next level of speaking, that of *concrete speech,* is characterized by the ability to convey ideas accurately and specifically and to discuss events of a relatively concrete nature. Included in the term "concrete" are things which are familiar, personal, visible, palpable, or repeated. At this level a discourse by the patient would include relating specific details of the day's events, an accurate description of physical symptoms, or a detailed account of important factors in his job. Most aphasic patients make frequent errors of speaking when required to perform at this level. Other patients, unable to perform at a more abstract level, are able to respond when the concepts are converted to a more concrete level. Illustrative of this is the patient who was unable to answer the question, "What does one do with money?" However, this was converted to the concrete, personal, and familiar experience by changing the question to "What does your wife do with money?" His response came promptly with bell-like clarity, "She spends it."

The highest level of speaking is the ability to communicate high-level concepts clearly by abstract speech. Discussion of international events, the duties of a good citizen, and the meaning of proverbs are examples of this level of speaking. An example of an aphasic response at this level occurred in answer to a request to explain the meaning of "Don't change your horses in midstream." The response demonstrated aphasic circumlocution and followed in this manner: "You should go along and cover your condition before you try to change in the last minute what you are doing." At this level of performance it may be difficult to distinguish between aphasia and general impairment of intellectual functions in some patients.

In general terms, the severer the aphasia, the lower the level of speaking in the described continuum. The actual phenomenon ob-

served in an aphasic patient will depend on the basic defect and on the way the patient compensates for the defect. There appear to be two basic defects in aphasic patients, namely, blocks and distortions.

Blocks are more or less transient absences of speech. They seem to represent an elevation of the threshold for the speaking of the specific word desired. In undisguised form, blocks appear as slowness, pauses, hesitations, and, rarely, as stuttering. A patient may compensate for blocks by the use of starters ("itsa," "well," "a-ah"), parenthetical asides ("oh, I should know that," "we have one of those at home") or the use of associations ("it's to write with, a pen, a pencil"). Another method of compensation is the substitution of another word for the desired word. The substitution may be accurate enough to convey the correct meaning or it may be inaccurate and fail.

Distortions of the events of speaking furnish the second basic defect of speaking in aphasic patients. Distortions of speech sounds affect most the sounds that require fine co-ordination of tongue and lip movements; this gives a defect which may closely resemble the usual forms of dysarthria. Words rather than individual sounds may be distorted. One patient expressed his exasperation at being examined with the following distorted but recognizable words, "Pull a cenpil out, drive me prazy." Distortions may involve phrases, as with the patient who defined courage as "gotta person be brave." Many times the distortions are mild enough to permit understandable speech and they may be corrected by the patient. One patient was asked, "What do you use to eat soup?" The progressively correct reply was "soup—snoup—spoup—spoon." When distortions are severe and uncorrected, and when they result in unrecognizable words or phrases, the condition is known as "jargon."

The speaking defects observed in aphasic patients increase when the level of abstractness, the amount of retention, or the specificity of response required is increased. Patients attempt to compensate for these problems by converting the response to a more concrete and personal level, by shortening the response, and by giving an approximate reply.

Previous words or concepts often intrude and interfere with speaking, giving rise to the phenomenon known as "perseveration."

The defects of speaking also will be modified by the social situation of the moment. The patient often talks better and easier with close members of the family than with strangers. One aphasic patient commented, "I can talk better one person with." A calm unhurried reassuring approach by the examiner encourages better performance by the patient. Conversely, placing the patient "on the spot" will increase his aphasic difficulty.

Defects of other language modalities will be seen in almost all aphasic patients. Defects of writing generally parallel those of speaking. A description of defects of listening is beyond the scope of this article. However a few comments seem worth including. There seems to be a basic defect in the recognition of individual words with severe listening defects and difficulty in comprehending the meaning of phrases and statements in milder defects. Some patients have commented that the sounds of words seem distorted. These basic defects of listening may produce some resemblance between the aphasic and the hard-of-hearing patient. In addition to the defects described there is also impairment of retention of things which have been heard. Many aphasic patients are able to interpret a general social situation and mask their receptive defect. Unless listening is tested specifically, the physician may fall into the belief that the defect is entirely expressive. Defects of reading appear to follow patterns similar to defects of listening.

A few words concerning prognosis in aphasia seem appropriate. Before attempting to estimate prognosis, the underlying neurologic lesion must be reasonably stable. Although spontaneous improvement may continue for much longer, sufficient stability will have occurred within 3 months after a nonprogressive lesion has been diagnosed to permit a reasonable estimate of prognosis. The occurrence of severe neurologic deficits other than aphasia is commonest among patients with severe aphasic defects. Only the rare aphasic patient has minimal neurologic defects or no neurologic defect other than aphasia. A persistent, severe defect of listening is indicative of a poor prognosis. Lack of motivation or evidence of general intellectual impairment imposes limitations on recovery. Detailed testing is necessary to establish an accurate prognosis. Such testing and therapy for aphasia are properly the function of speech pathologists with special training in aphasia.

MAYO CLINIC

REFERENCES

1. Nielsen, J. M.: Agnosias, Apraxias, Speech, and Aphasia. In Baker, A. B.: Clinical Neurology. Paul B. Hoeber, Inc., New York, pp. 352-378, 1955.

2. Wepman, J. M.: Recovery from Aphasia. Ronald Press Co., New York, 1951.

3. Brown, J. R., and Simonson, Josephine: A Clinical Study of 100 Aphasic Patients. I. Observations on Lateralization and Localization of Lesions. Neurology 7:777-783, 1957.

4. Penfield, Wilder, and Roberts, Lamar: Speech and Brain-Mechanisms. Princeton University Press, Princeton, New Jersey, 1959.

XCVII

THE USE OF REGIONAL FLAPS IN HEAD AND NECK SURGERY

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The concept of neck replacement by regional flap transposition in instances where the risk of healing has been seriously handicapped by a devitalized tissue bed has much to recommend it. This concept should be an integral part of the pre-operative plan, including adequate flap replacement as a part of the composite resection in radical extirpative operations of the head and neck. This should be so regionally situated that it can be immediately transposed into the deficient neck wound as a continuance of the primary operation. This has improved the rehabilitation of these precarious wounds and added increased security to the operative technique. The technique has been developed over the past 15 years to include various types of tissue flap replacements to different deficiencies in unique positions in the neck in both the male and the female. These deficiencies relate to severe post-irradiation status, fungating metastatic cancers and pharyngostomes and esophagostomes.

In the past many seriously damaged wounds would undergo primary mucous membrane and skin repair with very little chance of healing. The postoperative course was frequently complicated by tissue slough, secondary infection, fistulization and often fatal carotid artery hemorrhage.

A pre-operative analysis of tissue-bed quality in the operative field is essential for proper planning. The most frequent and most serious type of tissue devitalization in the neck is the result of irradiation. The majority of the individuals in this status have undergone

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some type of intensive irradiation for a primary cancer in the oral cavity, hypopharynx, larynx, cervical esophagus or thyroid. Some of these individuals may have had intensive irradiation for metastasis to the cervical lymph nodes. Other individuals present with a history of having had radiation treatment for benign lesions of the thyroid, tuberculous glands of the neck, tonsillitis, papillomata, and for such conditions as acne, hypertrichiasis and various dermatological conditions. One of the prime considerations under these circumstances is an evaluation of the warranty for an extensive operation in this type of tissue bed. The intrinsic condition of the surface of the neck may range from mild atrophy, telangiectasia and fibrosis to a "frozen neck," where fibrosis, atrophy and diminished vascularity have reached critical proportions.

The general value of tissue resurfacing in the neck is to add an adequate healthy epithelial flap covering and supply protection to the common and internal carotid arteries. The acquisition of this tissue in the neck also facilitates tubular reconstruction of the gullet and/or may be used as a pedicled tissue flap over a newly formed gullet.

INDICATIONS

There are multiple situations requiring the use of regional flap transposition to the neck:

1. Radical neck dissection alone when the tissues of the neck have been irreparably damaged by irradiation and the primary cancer is controlled; these necks are firm, fibrous, atrophic and semi-fixed and do not have a reasonable chance of healing per primam when the neck flaps are repositioned. It is unreasonable to expect this quality of tissue to heal following radical surgical intervention when its biological capacity has been so seriously devitalized. The pre-operative planning of the resection of this devitalized tissue, and its replacement by an immediate rotated chest flap, is imperative.

2. Radical neck dissection where metastatic cancer has fungated through the skin and the primary cancer has been controlled by surgery or irradiation (irradiated or non-irradiated tissue bed): a wide epithelial surface resection is necessary and there is no choice but to plan pre-operatively appropriate regional tissue flaps to resurface the wound.

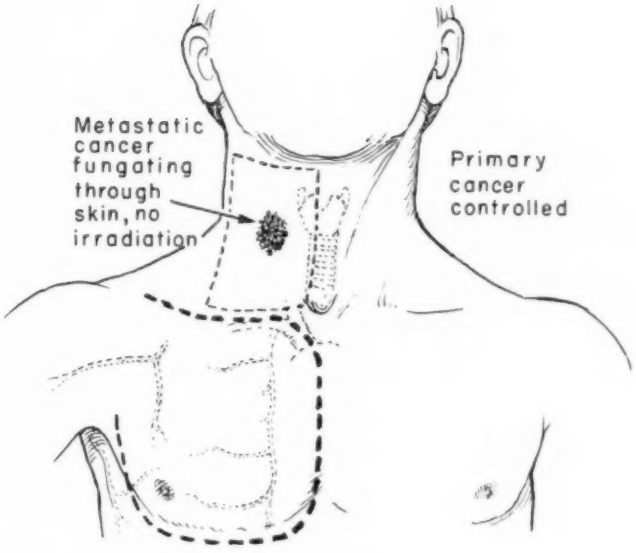


Fig. 1.—Radical neck dissection only, where metastatic cancer has fungated through skin. Primary cancer controlled. One-stage resurfacing procedure.

3. Composite extirpative operations in non-irradiated cases where the primary cancer in the oral cavity, pharynx, larynx, or cervical esophagus has not been treated and where the metastasis has compromised the skin of the neck: in a non-irradiated tissue bed one is justified in attempting a primary repair of the gullet if adequate mucous membrane remains, with immediate resurfacing by a regional tissue flap as a one-stage procedure.

4. Composite extirpative operations in heavily irradiated cases where skin may or may not be compromised: the increased risk of failure of primary healing of the post-irradiated mucous membrane tissues makes primary repair hazardous, frequently complicated by a breakdown of the mucous membrane suture line, fistula formation, secondary infection, protracted morbidity and possible carotid artery hemorrhage. Preferably, an elective pharyngostome, utilizing neck

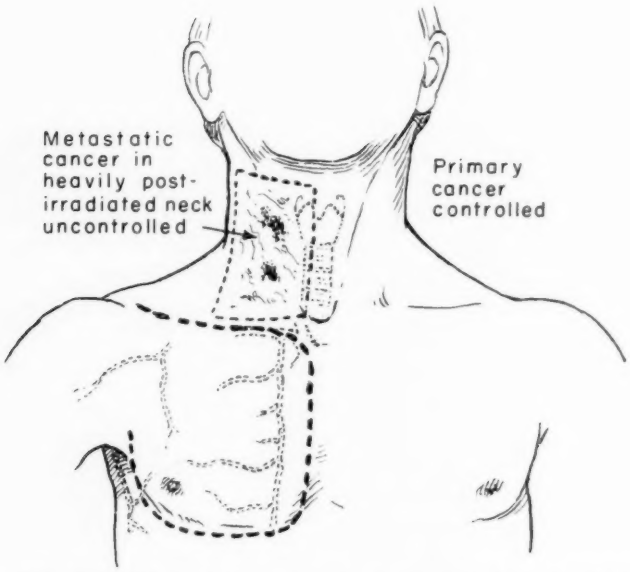


Fig. 2.—Radical neck dissection only, in compromised post-irradiated neck. Primary cancer controlled. One-stage resurfacing procedure.

flaps or chest flaps for covering the carotid artery is employed. The pharyngostome is then closed in an interval of one to two months as a secondary procedure by inverting the residual pharyngeal mucosa or neck skin, and then utilizing an immediate chest flap for resurfacing the new gullet.

5. Closure of pharyngostome problems when neck skin is used to create a new gullet but is inadequate for resurfacing: these situations include composite resections with unilateral neck dissection where the post-irradiation status prevails. Under these circumstances enough tissue remains in the neck to create a gullet, but the epithelial resurfacing of this new gullet is best accomplished by regional flap transposition. Composite operations with bilateral radical neck dissection in the normal, or post-irradiated wound status, create a problem where it is impossible to completely rehabilitate the gullet by neck flaps alone. A new gullet is formed by the inversion of the adjacent tissues

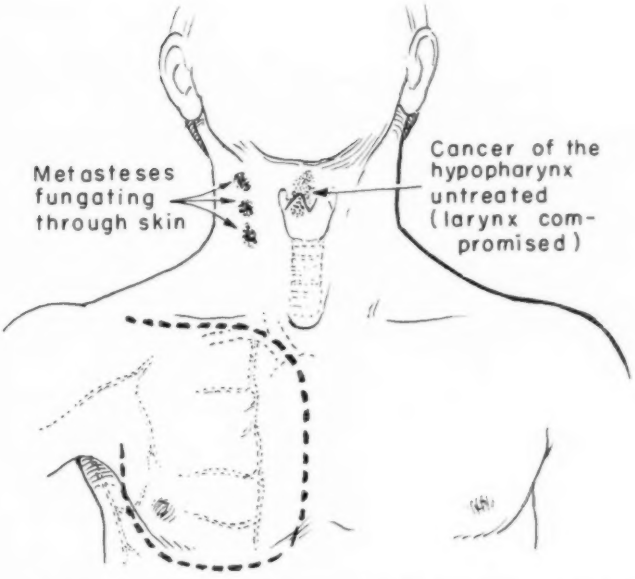


Fig. 3.—Composite resection, untreated, skin compromised. One-stage resurfacing procedure.

of the neck, but there is never adequate residual tissue in the neck to resurface this new gullet. Resurfacing is accomplished by regional flap transposition.

TYPES OF FLAPS

It should be emphasized that these flaps are not delayed in their transference, but are transplanted immediately in a one-stage technique. This single fact gives the regional flap its importance and distinction from distant and delayed flaps. The employment of distant pedicles from the abdomen, arm, chest, back, etc., formed into tubes and then over a period of months migrated into the wound and then remodelled after implantation, presented the patient with such a protracted morbidity and multiphase surgical experience that this in itself was often considered a deterrent to the advisability of an

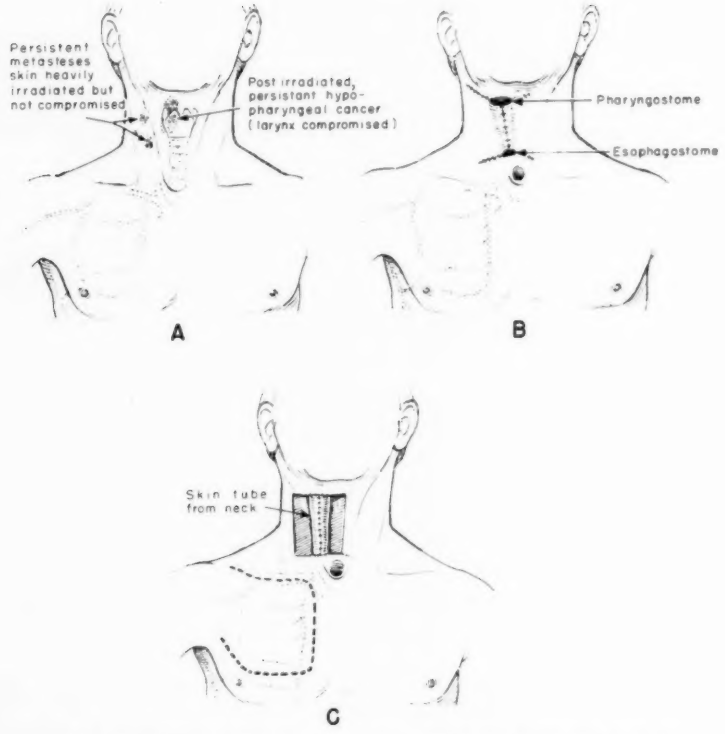


Fig. 4.—Composite resection, post-irradiated status, unilateral neck dissection. Two-stage rehabilitation procedure.

extensive primary resection. These protracted procedures have very little place in head and neck surgery today.

There are basically five types of regional flaps which are applicable for resurfacing maneuvers in the neck. These various flaps have particular and specific advantages relating to their regional blood supply, their local position in relation to the wound, the size of the wound, the presence or absence of local irradiation, the phenomenon of hair bearing coverings, and the variations in chest physiognomy in the male and female.

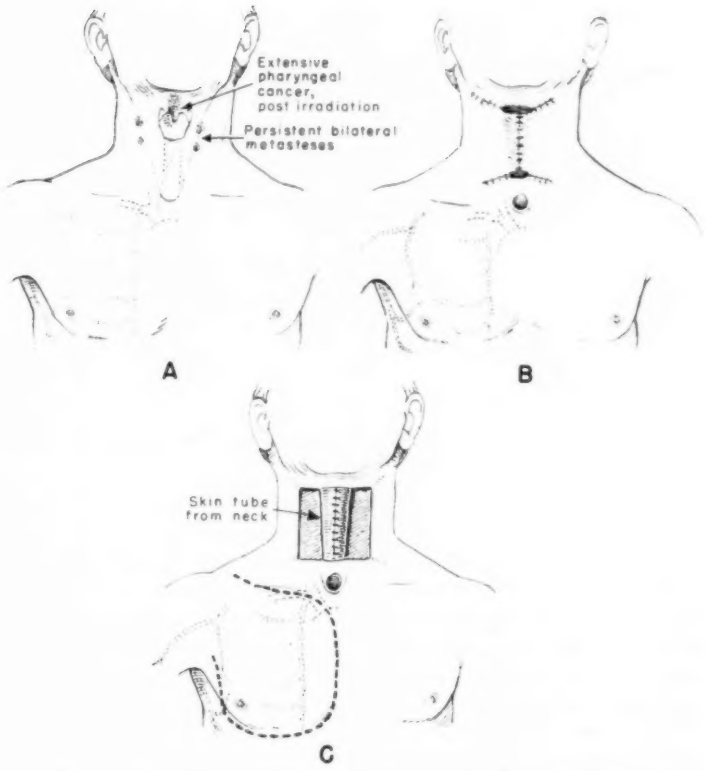


Fig. 5.—Composite resection, irradiated or non-irradiated, bilateral neck dissection. Two-stage rehabilitation procedure.

1. The most utilitarian flap used in resurfacing the neck is the anterior chest flap with the lateral pedicle. The vast majority of the primary cancers in this category requiring this special management occur in males. This flap has limitations in the female because of the size and position of the female breast and the factor of breast mutilation. The anterior chest wall is rarely included in the field of irradiation and contains little hair. Usually only unilateral pre-operative planning is required; however, the use of bilateral anterior chest flaps

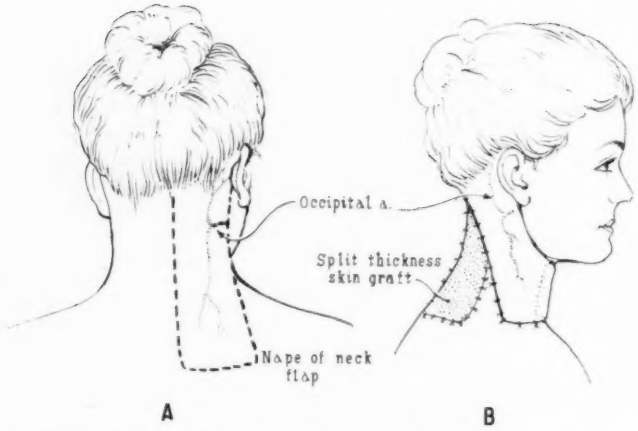


Fig. 6.—Nape of neck flap suitable in females, non-irradiated. One-stage rehabilitation procedure.

has been occasionally necessary. The size and the style of the flap depends naturally upon the requirements of the wound in the neck. Deficiencies in the lower part of the neck require only small flaps from the upper part of the chest, whereas resurfacing the entire lateral neck requires rectangular flaps measuring 12 x 20 cm and including the nipple and male breast. These flaps depend upon their arterial circulation from the branches of the thoraco-acromial artery, axillary artery, lateral thoracic artery and deltoid artery. The flap includes skin, subcutaneous tissue, fat, and a thin layer of fascia overlying the pectoralis muscles.

2. The anterior chest flap with the medial pedicle is particularly adapted to deficiencies in the lower part of the lateral or central neck. This flap ordinarily cannot be tailored as large as the laterally pedicled chest flap and will consequently not reach to the upper limits of the neck. Again, the absence of hair and the unlikelihood of this having been previously irradiated and its proximity to supraclavicular and suprasternal deficiencies gives it significant value. It also may be planned on unilateral or bilateral basis. Its essential blood supply is derived from the perforating branches of the internal mammary artery.

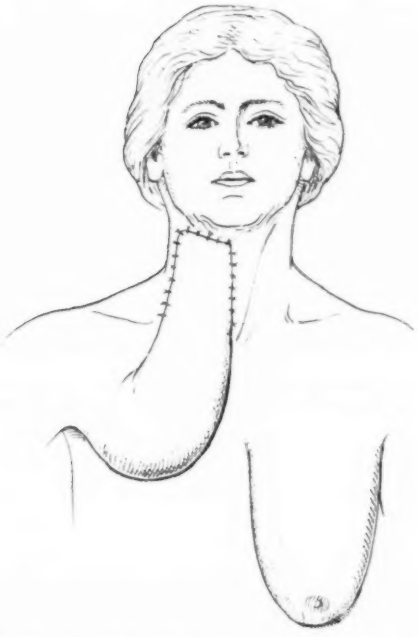
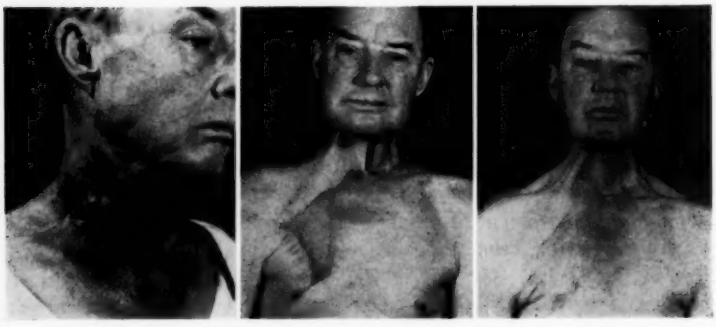


Fig. 7.—Breast pedicle. Two-stage rehabilitation procedure.

3. The nape of the neck flap with the superior subnuchal pedicle has proven to be of great value in resurfacing the neck in females. This flap is not delayed. It contains skin, subcutaneous tissue and fat, and the fascia over the trapezius muscle. It is wrapped around the neck like a muffler, leaving an inconspicuous donor site posteriorly and matching the naturalness of the neck well in color and consistency. Some have extended beyond the midline of the neck anteriorly and have measured 21 cm in length. This flap depends upon its essential nourishment from the postauricular artery and the occipital artery. If these arteries are inadequate, the flap is compromised. These flaps ordinarily contain little hair in the male or female. The principal disadvantages are that they are positioned posteriorly in the region of the nape of the neck and are awkward to mobilize and transpose. A biological deterrent is that these flaps are frequently included in or

Fig. 8.—Post-surgical, post-irradiation failure with recurrent neck metastasis treated by re-resection of neck and immediate resurfacing with rotated chest flap.



A B C

Fig. 9.—*A*, recurrent post-irradiated, post-operated cancer of thyroid with metastasis; *B*, post-thyroidectomy, -laryngectomy, -bilateral neck dissection; neck resurfaced with chest flap at same operation; *C*, rehabilitation of gullet.

approximate the field of irradiation for the primary cancer in the head and neck. Thus, a certain amount of post-irradiation effect may have altered the viability of the flap. This is considered a contra-indication to the use of the flap. There are modifications in the design and styling of the flap with its positions over the lower part of the neck and scapula that are suitable for special problems.

4. Scalp flaps have particular applicability to deficiencies in the upper part of the neck, about the ear, parotid gland and subdigastric areas. Scalp flaps are hair bearing flaps, except in alopecia, and are consequently best adapted to the male. The flaps are usually patterned in the occipital or parietal areas and utilize th perforating branches of the occipital artery, postauricular artery and superficial temporal artery.

5. The female breast may be used as a pedicle flap for the lower neck under specialized circumstances. If the nape of the neck flap or anterior chest wall flap is precluded and if the breast is thin and sufficiently long, it may be immediately transposed onto the lower part of the neck for resurfacing purposes. In this particular instance, it is necessary to release the pedicle after an interval of three or four weeks and tailor the breast tissue on the neck and return the residuum of the breast to the chest. This technique is rather curious but has a limited practical value.

TECHNIQUE

The dominant feature of the technique is that the flap is immediately raised and transposed. At this stage, it is soft, supple and flexible. It is sutured into the wound in layers without tension or distortion. Particular care should be taken to eliminate hematoma and saliva from the wound. Only slight pressure is applied to the pedicle flap with the dressing. If there are any signs of insufficient arterial supply or venous return, reposition of the flap into its original bed is mandatory. The flap may then be transposed two weeks later. There are many circumstances where this is not practical, namely when one is committed to cover the essential vascular structures of the neck with a regional flap at the primary operation. Under these circumstances, some type of modification of the flap, or the use of two flaps in conjunction with a free skin graft must be considered. If

the nipple is in a conspicuous position on the neck, it may be excised as a minor office procedure at a later date.

Slough of the nipple has never been observed. The flaps heal per primam in almost all instances. Very occasionally a 1 cm eschar may form superficially at the extreme periphery. There are "dogears" at the posterior inferior portion of the flap that may require minor modifications. The donor area on the chest or at the nape of the neck or scalp is dressed with a thick-split free skin graft held in position over a bolus.

SUMMARY AND CONCLUSIONS

1. Regional pedicle flaps offer the most efficient source of covering for deficiencies in the neck following radical neck surgery associated with fungating metastasis or severe post-irradiation status with compromise of the skin.

2. These flaps should be planned as an integral part of the primary excisional operation.

3. Their positions are on the anterior chest, the nape of the neck and the scalp.

4. Their great advantage over other techniques is that they can be moved without delay in a single stage procedure as a continuance of the primary operation.

5. They shorten morbidity and add surgical security to the operation.

6. They accomplish resurfacing, protect the carotid artery system, and add tissue for rehabilitation of the gullet.

139 East 36th Street

Abstracts of Current Articles

EAR

Audiogram of I.B.M. (International Business Machine) Operators and Analysis of Noise of I.B.M.

Ebihara, I., Harigaya, M., Motosugi, M., and *Yatomi, R.*: J. Oto-rhino-laryng. Soc., Japan, 62:2020 (Sept.) 1959.

Hearing tests were done on seventeen I.B.M. operators together with the analysis of the noise of four kinds of machines. So-called C^5-dip was detected only in those using the accounting machine. And then a characteristic spectrum specific for the accounting machine was obtained in the noise analysis.

It appeared that of the I.B.M. machines tested only the noise of the accounting machine induced an occupational deafness.

The author concludes that occupational deafness in the workshop may develop in factories where the surrounding noise is not loud.

HARA and OGURA

A Method of Bone Conduction Measurement with Gradually Increasing Masking Noise

Takita, Z.: J. Oto-rhino-laryng. Soc., Japan, 62:1697 (Aug.) 1959.

There is still a controversy about standardizing the method of bone conduction audiometry. This is because of the difficulty of masking the shadow hearing in the opposite ear.

In this article, the author advocated the use of gradually increasing masking noise as a better method of masking in the test of hearing by bone conduction. This method has the following advantages.

1. No need to settle which ear should be masked before testing.

2. No influence by plug effect with masking receiver applied to the contralateral ear.

3. No need of consideration about the masking effect of the noise.

4. Least mental influence of the examinee.

5. Valuable for the differential diagnosis of various cases in which the masking was ineffective.

HARA and OGURA

Hereditary Studies on Congenital Atresia of the Ear

Hayasaki, H.: J. Oto-rhino-laryng. Soc., Japan, 62:1835 (Aug.) 1959.

Of 144 patients with congenital atresia of the external auditory canals the author made systematic studies in 98 families, in which there was a hereditary history.

The morbidity was 2.02% among patients' fathers and 0.61% among their aunts. These morbidities were higher compared with 0.0016% of those of the general population. The same condition was found in twin brothers.

The sex ratio was 78.49% in the male and 21.51% in the female, and 30.63% on the left, 63.96% on the right and 5.41% on both sides. These ratios were similar to those reported by American, European and other Japanese authors.

When one congenital malformation is investigated several associated malformations and congenital anomalies are found in the families.

In this study 18.18% of malformation was found in patients' fathers and 29.9% in their families. These abnormalities included: 1) congenital atresia auris, 2) congenital paralysis of the facial n., 3) congenital lameness, 4) congenital scoliosis, 5) microphthalmus congenita, 6) polydactylia and 7) congenital deaf-mutism.

HARA and OGURA

Studies on PGSR Audiometry

Kanadani, M.: J. Oto-rhino-laryng. Soc., Japan, 62:1951 (Sept.) 1959.

The early and accurate evaluation of hearing impairment of children is becoming more and more important in the field of otology, especially from the viewpoint of prognosis.

Hearing tests in young children are divided into two main groups: PGSR audiometry and play audiometry. Also, there are some simpler tests, such as startle reflex test.

The author devised an apparatus for recording the psychogalvanic skin response. He took the records of examination on normal and hard of hearing children by PGSR audiometry with various tones ranging from 500 to 4000 cps. The results showed that this method was reliable as the objective hearing test.

The author compared this method with play audiometry and he obtained the following results.

PGSR audiometry was successful in 16 children out of 23 in the age range of one year and nine months to 5 years and eleven months. Successful results with play audiometry were obtained in 13 children out of the group of 23 in the age range of 3 years and 6 months to 5 years and 11 months.

PGSR audiometry has been proved to be a more satisfactory test for younger children than play audiometry. Compared with play audiometry or startle reflex test, the results were considerably more reliable and it was an excellent method for the examination of hearing acuity in younger children with severe hearing loss, who are extremely difficult to test by other methods.

HARA and OGURA

A Study on the Measuring Method of the Impedance of the Ear Drum

Miyano, K.: J. Oto-rhino-laryng. Soc., Japan, 62:1980 (Sept.) 1959.

The impedance of the living human eardrum was measured following Troger's method. The results were analyzed in detail experimentally and theoretically.

The value of the impedance obtained by the author was found similar to those of Troger, Geffcken, Waetzmann and Metz. It differed from that of Zwislocki.

The impedance was higher in a lower frequency range and lower in a higher frequency range. Finallly, the author measured the impedance of a rubber membrane by Troger's method. By the author's method this was not applicable to the human ear but most exact in measuring acoustical impedance. A comparative study of these two results showed that the impedance obtained by Troger and others was not correct. A more correct method for the measurement of the impedance of the human ear drum should be developed in the future.

HARA and OGURA

Study on Mycotic Infection of the External Ear Canal

Zaoli, Giancardo: Bollettino delle Malattie dell'orecchio, della Gola, del Naso (July) 1959.

The author reports a series of 59 cases of external ear infections seen at the University Clinic in Perugia. The cases were checked daily in the clinics. Cultures were made of the secretions and the tissues obtained from the external canal were examined in the various stages of the disease.

In this series of 59 cases, 44 were found to have mycotic infections. In this preliminary report Zaoli divides external ear infections into two stages. The first stage is the appearance of a bacterial infection. The resulting edema prepares the tissue for the second stage: if and when the bacterial flora disappears, mycotic infection follows. The first stage may last from four to ten days. The second stage has an indefinite time period.

The author concludes from his examinations that otomycosis begins as a bacterial infection which is followed by a mycotic infection in the second stage. Local antibiotics did not help the mycotic infection. This accounts for some of the unexplained failures encountered in the local therapy of external ear infections.

MURGOLO

A Further Report on the Chorda Tympani Syndrome

Young, A. G., and Stein, G. E.: Brit. M. J. 1:620-621 (Feb. 27) 1960.

A patient presenting a syndrome of unilateral sweating and flushing of the submental region following eating is described. As it was felt the impulses traveled over the chorda tympani, this nerve was sectioned in the middle ear. The abnormal sweating was instantly relieved but began to recur in six months. Feeling some regeneration had occurred, the operation was repeated removing a portion of the nerve. Immediate improvement lasted one year; the attacks then returning rarely and not severely. The similarity to the auriculotemporal syndrome is discussed. If this syndrome proves to be a definite entity, they feel the endaural operation as described should have merit.

TRIBLE

The Ventriculomastoid Shunt

Lake, S. F.: Laryngoscope 69:1469-1477 (Dec.) 1959.

A modification of the Nosik procedure for shunting the cerebrospinal fluid from the lateral ventricle to the mastoid antrum, was performed in 54 patients between 1949 and 1955. These included 18 with congenital hydrocephalus, 17 with increased intracranial pressure without brain tumor and over 19 for increased intracranial pressure with intracranial tumor.

Although the procedure carries with it some complications, it can be performed successfully as a life saving procedure under local anesthesia in the very ill patient. Revisions become necessary because of obstruction of the mastoid end of the shunt by granulations or displacement of the tube. Meningitis was a complication which had to be contended with in the immediate as well as the late postoperative period.

SENTURIA

Changes in the Vestibular Labyrinth with Intense Sound

Mangabeira-Albernaz, P. L., Covell, W. P., and Eldredge, D. H.: Laryngoscope 69:1478-1493 (Dec.) 1959.

Evidence is presented that in guinea pigs allowed to survive up to 133 days, there was damage to the vestibular labyrinth accompany-

ing the severe cochlear changes caused by exposure to single frequencies varying from 150 to 50,000 cps. The most common alteration was rupture of the partition separating the utricle and semicircular canals from the saccule and the remainder of the vestibule. The walls of the saccule and utricle were ruptured and collapsed in a small percentage of the cases and to a degree, rupture and collapse was correlated with the frequency of the traumatic tone. The sensory and supporting cells of the maculae of the saccule and utricle showed no change.

SENTURIA

Experimental Studies on the Variation of Skin Resistance and the Stimulation of Human Labyrinth

Kikuchi, J.: J. Oto-rhino-laryng. Soc., Japan, 62:1721 (Aug.) 1959.

In order to determine the relation between sweat glands and labyrinthine reflex the author observed the change of skin resistance by labyrinthine stimulation in the human body.

Sink resistance after the labyrinth stimulation showed a reduction to as much as 1/3 to 1/8 of the average level before stimulation. It seemed that this decrease mainly depended on the decrease of reactivity.

The degree of skin resistance after labyrinth stimulation fluctuated in each case when adrenalin, pilocarpin, atropin, tebron and immidarin injections were given preceding stimulation.

The change of skin resistance by labyrinth stimulation seemed to be closely related with the excitement of sympathetic cholinergic nerves which control sweat glands.

HARA and OGURA

NOSE

Optic Neuritis and Frequency of Sinus Infections

Dorello, U., Scardovi, C.: Rivista Oto-Neuro-Oftalmologica (Oct.) 1959.

The authors give a statistical report of the frequency which sinus infections are met with in optic neuritis. The series consisted of 279

patients; of this number 127 were traced to paranasal sinus disease. Optic neuritis was divided into three pathological entities:

a. Retrobulbar neuritis - an inflammation of the orbital position of the optic nerve - these cases had 62% sinus involvement.

b. Papillitis - inflammation of the optic disc. These cases showed 50% sinus involvement.

c. Atrophies - post-neuritic type, especially after prolonged papilledema - these cases showed 33% sinus involvement.

The frequency of involvement of the various sinuses,

a. Maxillary sinus; most frequently

b. Frontal sinus; next in frequency

c. Ethmoid sinus; next in frequency

d. Sphenoid the least common.

Treatment consisted of proper surgical drainage of sinuses involved, plus antibiotic therapy.

The authors found that sinus infections are a more frequent cause of optic neuritis than has been previously reported in the literature.

MURGOLO

Roentgen Features of Nasal Accessory Sinus Mucoceles

Palubinskas, A. J., and *Davies, Hugh*: Radiol. 72:576 (Apr.) 1959.

The authors discuss the clinical and roentgen features of nasal accessory sinus mucoceles and present 13 cases. A short clinical history, radiographic features, operative findings and postoperative course is given for six cases. Clinical and roentgen features are given in seven additional cases.

They discuss a definition, the occurrence, etiology, the natural history of the anatomical development, clinical findings, and pathol-

ogy of accessory sinus mucoceles. Other conditions in this area to be considered in the differential diagnosis and treatment are discussed. Eight radiographs and 34 references are included.

JORSTAD

The Systematic Use of Tomography in the Diagnosis of Carcinoma of the Paranasal Sinuses

Dodd, Gerald D.; Collins, Lois C.; Egan, Robert L.; and *Herrera, J. Raul*: Radiol. 72:379-391 (Mar.) 1959.

The authors have examined, by tomography, 123 patients with primary and 68 with secondary tumors of the ethmoid, maxillary or sphenoid sinuses. No malignant frontal sinus lesion was encountered. Tumors occurred twice as frequently in males as in females. The majority arose in the maxillary antra. Seventy-three per cent of the primary neoplasms were squamous carcinoma. The majority of malignant tumors of the parnasal sinuses are indolent by nature.

The roentgen diagnosis of malignancy of the paranasal sinuses depends upon the demonstration of bone destruction. Experiences with these 191 patients have caused the authors to plan great dependence upon tomographic studies in the earlier diagnosis and management of the lesions. Tomography is essential in the detection of minimal bone destruction in the area. Tomography provides reliable information as to extent of the disease. Eight references, a chart showing histology and location, a tomographic classification, 11 radiographs and 12 tomographs are included.

JORSTAD

LARYNX

Morphologic Changes of the Larynx and Inferior Resonant Cavity in Phonation, Studied by Means of High-Voltage Radiography

Tsuboi, H.: J. Oto-rhino-laryng. Soc., Japan, 62:2064 (Sept.) 1959.

Using 25 persons including eight vocalists, both sagittal and lateral high-voltage radiograms were taken in order to study morphologic changes of the soft parts of the larynx and the pharyngeal cavity in phonation. Tsuboi observed the changes produced in various pitches, registers, forces and vocals.

1) The ventricle of larynx showed the most conspicuous changes. The higher the pitch within the same register, the narrower the ventricle. In the chest voice register, however, the ventricle turned from narrow to wide and again to narrow as the pitch rose. At the same pitch, the ventricle proved always wider in the higher register.

2) Within the same register, the vocal cords were elongated as the pitch rose. But in the male falsetto voice register and the female head voice register, the length of the vocal cords remained constant. At the same pitch, the vocal cords did not differ in length in two different registers.

3) The higher the pitch within the same register, the higher the site of the larynx. At the same pitch, no definite characteristics were noted of the site of the larynx in two adjoining registers.

4) Individual differences were noted to such an extent in the relation between the width of the pharyngeal cavity and the changes in pitch or register, that specific relationship was not observed.

5) The width of the ventricle of the larynx and the pharyngeal cavity changed most remarkably in accordance with changes in force.

HARA and OGURA

MISCELLANEOUS

An Experimental Study of the Suture Method of a Fine Motor Nerve

Morita, H.: J. Oto-rhino-laryng. Soc., Japan, 62:2086 (Sept.) 1959.

The repair of an injured nerve is one of the essentials in modern surgery. The suture of injured nerves, however, is so complicated that the results obtained are often unsatisfactory, especially in the otolaryngological field.

The author devised a new surgical method experimentally for the end-to-end repair of injured facial nerves using guinea pigs. The principle of the method consisted in twining the pericapsular connective tissue of the nerve without using any suture material so as to bring the ends of the nerve into contact with each other, and then the injured portion was fixed with surrounding tissues.

After the operation the nerve axis was observed to regenerate from the proximal end at the rate of 0.2 to 1.0 mm a day, and regenerate completely within 12 to 16 weeks. The author suggests that the operation should be done as early as possible after the injury to get a satisfactory result.

To this end Morita states that the material used for repair must be easily available. There should be no serious foreign body reaction and repair must be accomplished without injuring the nerve axis with the suture material.

HARA and OGURA

Investigations on the Hemorrhage from Nose and Throat

Inoue, H.: J. Oto-rhino-laryng. Soc., Japan, 62:1737 (Aug.) 1959.

In otolaryngologic practice the bleeding tendency signifies that either the patient has a spontaneous propensity to bleed profusely or the practitioner finds bleeding which is hard to control after an operation.

Inoue states that testing of the mucous membrane itself was better than testing of the skin as a method to determine the bleeding tendency of the mucous membrane.

The bleeding tendency in cases with spontaneous and postoperative bleeding from the nose and pharynx was investigated.

1. In spontaneous bleeding, vascular fragility played a most important role. On the other hand, coagulability of the blood was more important in the operative and traumatic bleeding.

2. Among the systemic hemostatics, those which acted on the blood vessels were most effective. "Thromboplastin" was most effective for the blood coagulation.

HARA and OGURA

Radical Pre-operative Roentgen Therapy in Primarily Inoperable Advanced Cancers of the Head and Neck

Buschke, Franz, and *Galante, Maurice*: Radiol. 73:845 (Dec.) 1959.

Ten cases of inoperable carcinoma of the head and neck are presented. The lesions represented differentiated epitheliomas of the

pillar, palate, floor of mouth and gingiva of a type that can be expected to infiltrate locally or to remain limited to the regional lymphatic areas for a long time, but too extensive for complete removal by primary surgery and either too differentiated or too invasive into bone, muscle, or lymph nodes for control by radiation therapy alone.

Radiation therapy was given at one million volts through a single field. The dose varied between 6000 and 8000 r (skin) in 32 to 59 days for a minimal tumor dose of 4100 to 6500 r. Surgery was done from one to five and one-half months after completion of radiation. The interval, if possible, should not exceed two or three months because of the progressing vascular and fibrotic changes.

Between February 1957 and February 1958, the combined procedure was carried out in six patients. In three cases the surgical excision was far beyond histologically demonstrable disease, and in three no disease was demonstrated in primary area or lymph nodes.

In spite of the heavy pre-operative irradiation there were no surgical complications beyond those associated with this type of surgery. One operated five and one-half months presented more fibrosis than at one month but healing progressed satisfactorily.

The authors emphasize that these cases demonstrate that surgery and radiation are not competitive or mutually exclusive. The planned utilization of both disciplines can be of great help in the management of properly selected cases. A table of data for each case and two references are included.

JORSTAD

Books Received

Hearing and Deafness

By *Hallowell Davis, M.D.*, Director of Research, Central Institute for the Deaf; Professor of Physiology and Research Professor of Otolaryngology, Washington University School of Medicine; *S. Richard Silverman, Ph.D.*, Director, Central Institute for the Deaf; Professor of Audiology, Washington University. Pp. xii + 573. New York, Holt, Rinehart & Winston, Inc., 1960. ($10.00)

Eleven years following the original publication of this authoritative book, Drs. Davis and Silverman, with the collaboration of thirteen specialists in the fields of hearing, speech and deafness, present a thoroughly revised and up-to-date edition.

This volume is carefully written and edited so that the new developments of our knowledge and techniques in otology and audiology and desirable changes in terminology are incorporated in many chapters of the book. Careful reading of this definitive work reveals that in addition to a description of the important strides in audiology there are also included many of the newer medical concepts regarding diagnosis and therapy and some anticipated developments in surgical rehabilitation of hearing.

The otologist will be pleased with the simplified, concise description of the developments dealing with biophysics and physiology of the cochlea. The guidance offered regarding the non-medical rehabilitative aspects of the patient's treatment will be invaluable.

Although there can be no question regarding the desirability of emphasizing the use of precise audiologic terminology, we wonder whether the otologists will be willing to accept the limitations on the use of such terms as "hearing loss" and "deafness." We see no reason to substitute an artificially restricted definition of the word dysacusis for those impairments "which are not simple losses of sensitivity of hearing."

Despite this mild reservation regarding terminology, this reviewer believes that this superb resumé of our present knowledge of hearing

and deafness properly belongs on the desk of every student and practitioner of otology and audiology.

Tumores Malignos de la Region Etmoidomaxilar (Malignant Tumors of the Ethmoidomaxillary Region)

By *R. Poch Vinals*, Catedratico de Otorrinolaringologia de la Facultad de Medicina de la Universidad de Sevilla. Paper, 8vo. 95 pages, 62 illustrations. Madrid, Editorial Paz Montalvo, 1959. (In Spanish)

Cosmetic Surgery; Principles and Practice

By *Samuel Fomon, M.D.* Cloth, 8vo., 651 pages, 608 illustrations. Philadelphia, J. B. Lippincott Company, 1960. Price $27.50.

A detailed and comprehensive work on plastic surgery by an experienced and well known author. Approximately one-half of the book is devoted to regions involving the ear, nose and throat, and most of the rest to fundamental surgical principles of procedure. There is a chapter on cosmetic eye operations and a brief one on Thorax, Abdomen and Extremities. The illustrations are excellent.

Clinical Applications of Bronchology

By *Dezso Kassay, M.D.*, Professor of Bronchoesophagological Research, Temple University School of Medicine, etc. Cloth, 225 pages, 116 illustrations. New York, McGraw-Hill Book Company, Inc., 1960. Price $15.00.

A compact manual and guide on the subject, well written, and clearly illustrated. Besides a general documentation of some 100 publications the author draws heavily on his own experiences, most of them recorded in the foreign literature between 1947 and 1957.

Chirurgie der Verengungen der Oberen Luftwege (Surgery of the Narrowing of the Upper Air Passages)

By *Prof. Dr. Aurel Réthi* of the Laryngological Division of the Semmelweis-Rochus-Hospital, Budapest. Paper, 8vo., 119 pages, 166 illustrations. Stuttgart, Georg Thieme Verlag, 1959 (U.S. and Canada, Intercontinental Medical Book Corp., N.Y.). Price $6.70. (In German)

A brief ready-reference manual on the various obstructions and their correction. Extensive bibliography of limited practical value since the titles are not given, as is customary in most European listings.

Le Laryngologiste et les Données Actuelles du Traitement des Insuffisances Respiratoires Aiguës (The Laryngologist and the Current Thought on Acute Respiratory Failures)

By *P. Aboulker*, with the collaboration of *J. Lissac* and *O. Saint-Paul*, and comments by several others. Paper, 8vo., 508 pages, 103 illustrations and 2 color plates. Paris, Librairie Arnette, 1959. Price 60NF. (In French)

This is a substantial monograph on the life-saving methods in use today in combating anoxias of various types, with a look at the underlying physiopathology. In their introduction the authors describe the year 1932 as "une date cruciale" when T. C. Galloway reported three patients moribund with bulbar poliomyelitis, "tracheotomized and assisted by the iron lung. Two of these patients were saved while the death of the third appeared to have been due to a too tardy intervention." A section is devoted to special aspects of the problem.

Notices

VII INTERNATIONAL CONGRESS OF OTORHINOLARYNGOLOGY

The Seventh International Congress of Otolaryngology will take place July 23-29, 1961, at Paris. The sessions will be held under the presidency of Professor Maurice Aubry at the New Faculty of Medicine, Rue des Saints-Pères.

Dr. Henry Guillon, Secretary General

6 Avenue Mac Mahon, Paris XVII, France.

ACADEMY EMPLOYMENT REGISTRY

In order to facilitate the growth of academic ophthalmology and otolaryngology in both clinical and basic science in the various medical schools and associated hospitals and institutions, an Employment Registry for Scientists has been established by the American Academy of Ophthalmology and Otolaryngology. The Registry will serve to aid in the placement of candidates completing training who desire academic and investigative positions and the replacement of those vacating such positions.

THE AMERICAN COLLEGE OF ALLERGISTS

The American College of Allergists will conduct a graduate instructional course in connection with its Seventeenth Annual Congress, March 12-17, 1961, at the Statler Hilton, Dallas, Texas. For information write John D. Gillaspie, M.D., Treasurer, 2141 14th Street, Boulder, Colorado.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The annual meeting of the Georgia Society of Ophthalmology and Otolaryngology will be held at the General Oglethorpe Hotel, Wilmington Island, Savannah, Georgia, on March 2-3-4, 1961. Guest speakers in the division on otolaryngology will include Dr. Lawrence R. Boies, Minneapolis; Dr. Francis W. Davison, Danville, Pa.; and Dr. Theodore Walsh, St. Louis.

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from March 13 through March 25, 1961, under the direction of Paul H. Holinger, M.D.

Registration will be limited to fifteen physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

MOUNT SINAI HOSPITAL

In order not to conflict with the International Congress of Otolaryngology to take place in Paris in July 1961, the annual intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 7 to January 20, 1961, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, New York.

NEW YORK EYE AND EAR ALUMNI

The annual spring meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 17-19, 1961.

Symposia will be offered on Otosclerosis and Benign Lesions of the Larynx. There will also be a closed circuit television demonstration of surgical procedures.

In addition, there will be lectures on plastic surgery, paranasal sinuses and carcinoma of the head, neck and larynx.

Further information may be obtained by writing to Dr. John R. Finlay, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

UNIVERSITY OF PENNSYLVANIA

The University of Pennsylvania Graduate School of Medicine announces full-time courses in Otolaryngology, Bronchoesophagology and Laryngeal Surgery.

Courses in otolaryngology and bronchoesophagology are now divided into two semesters of four months each. During the first semester (October to January), emphasis is placed upon basic medical sciences. Cadaver endaural surgery of the temporal bone, rhinoplasty, laryngeal surgery and bronchoesophagology are given in the second semester (February to May).

These courses are recognized for credit toward qualification by the American Board of Otolaryngology.

Registration will be limited to fifteen physicians.

Graduate School of Medicine
36th and Hamilton Walk
Philadelphia 4, Pa.

ROYAL SOCIETY OF MEDICINE, LONDON

Programs for the sessions 1960-61 of the Section of Laryngology will be on the following subjects:

December 2—Is the removal of tonsils and adenoids necessary?

February 3—Recent advances in the treatment of intrinsic carcinoma of the larynx.

March 3—Chronic sinusitis in children.

May 5—Vasomotor rhinitis.

Those of the Section of Otology will be:

December 2—Intracranial complications of aural disease.

February 3—1. Problems of tympanoplasty. 2. Experience with pedicled temporal muscle flaps in radical mastoid and tympanoplasty operations. 3. Is myringoplasty worth while?

March 3—1. The pathological bases of some labyrinthine vascular disorders. 2. Meniere's syndrome.

May 5—The present position of stapes footplate surgery.

Members of the American Laryngological Association, the American Otological Society and the American Triological Society who will be in the United Kingdom at the time of any of these meetngs will be very welcome.

TEMPLE UNIVERSITY

A two weeks' continuous course in allergy is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and the Graduate School of Medicine of the University of Pennsylvania from February 27 to March 10, 1961. Tuition Fee: $175.00 . Enrollment limited.

For information please apply to

Dr. George Blumstein
c/o Temple Medical Center
Philadelphia 40, Penna.

UNIVERSITY OF TEXAS

The Post Graduate School of the University of Texas and the Baylor University College of Medicine will present a course in Allergy to be conducted by Herbert J. Rinkel, M.D., on January 30th through February 3rd in Houston, Texas.

The course will be followed by a meeting of the Gulf Coast Allergy Study Group on February 4th and 5th.

For further information, write to:

Office of the Dean
University of Texas Post Graduate School of Medicine
410 Jesse Jones Library Building
Houston 25, Texas.

UNIVERSITY OF TORONTO

On May 11, 12 and 13, 1961, there will be presented a graduate course by the Staff of the Department of Otolaryngology. They will be assisted by two distinguished guests: Dr. Philip E. Meltzer, Professor of Otolaryngology, Harvard Medical School, Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary; and Dr. W. G. Hemenway, Department of Otolaryngology, University of Chicago.

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in association with the Section of Otolaryngology of the Ontario Medical Association. The remainder of the sessions will be held in the Clinical areas of the University of Toronto.

The fee for the course will be $40.00 and will include a complimentary dinner.

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

The American Society of Facial Plastic Surgery will meet February 9 and 10, 1961, Hotel Peabody, Memphis, Tennessee, and June 28, 1961, Hotel Elysée, New York, N. Y.

UNIVERSITY OF MINNESOTA

The University of Minnesota announces a one-day continuation course in Otolaryngology for Specialists which will be presented at the Center for Continuation Study on January 26, 1961, preceding the Sectional Meeting of the American Laryngological, Rhinological, and Otological Society in Minneapolis on January 27 to 28, 1961. Guest registration for the meeting of the "Triological" will be welcomed. The program for the continuation course has been designed to complement the scientific program of the "Triological" Society.

Lodging and meal accommodations are available at the Center for Continuation Study.

ANNALS

In order to fulfill the requests for the March 1935, March 1937, March 1959 and June 1959 issues, the ANNALS will pay $3.50 for each book in good condition.

Please mail books to Zimmerman-Petty Linotyping Co., 2308 Olive, St. Louis 3, Mo.

OFFICERS

OF THE

NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: John H. Dunnington, M.D., New York
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, October, 1960

American Board of Otolaryngology

President: Bernard J. McMahon, M.D., St. Louis
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa
Examination: Chicago, Oct. 3-6, 1960

American Broncho-Esophagological Association

President: F. Johnson Putney, M.D., Philadelphia, Penna.
Secretary: Daniel C. Baker, Jr., M.D., 903 Park Ave., New York 21, N. Y.
Meeting: Lake Placid Club, New York, May 23-24, 1961

American Laryngological Association

President: Edwin N. Broyles, M.D., Baltimore, Md.
Secretary: Lyman G. Richards, Mass. Inst. Tech., Cambridge, Mass.
Meeting: Lake Placid, N.Y., May 21-22, 1961

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Theo. E. Walsh, St. Louis
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N.Y.
Meeting: Lake Placid Club, New York, May 23, 24, 25, 1961

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Lawrence R. Boies, M.D., Minneapolis, Minn.
Secretary, Walter E. Heck, M.D., Suite 103, 3905 Sacramento Street, San Francisco 18, Calif.
Meeting: New York City, June 26-30, 1961

American Otological Society

President: Henry L. Williams, M.D., Rochester, Minn.
Secretary-Treasurer: James A. Moore, M.D., New York Hospital, 525 E. 68th St., New York 21, N. Y.
Meeting: Lake Placid, N.Y., May 26-27, 1961

VII International Congress of Otolaryngology

President: Prof. M. Aubry, Paris
Sec. Gen.: Dr. H. Guillon, 6 Ave. Mac-Mahon, Paris XVII France
Meeting: Paris, July 15-22, 1961

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose Gros, Havana
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

Pacific Coast Oto-Ophthalmological Society

President: John F. Tolan, M.D., 1118 - 9th Avenue, Seattle 1, Wash.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah

Canadian Otolaryngological Society

President: Dr. Fernand Montreuil
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47 Mexico 7, D.F.

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